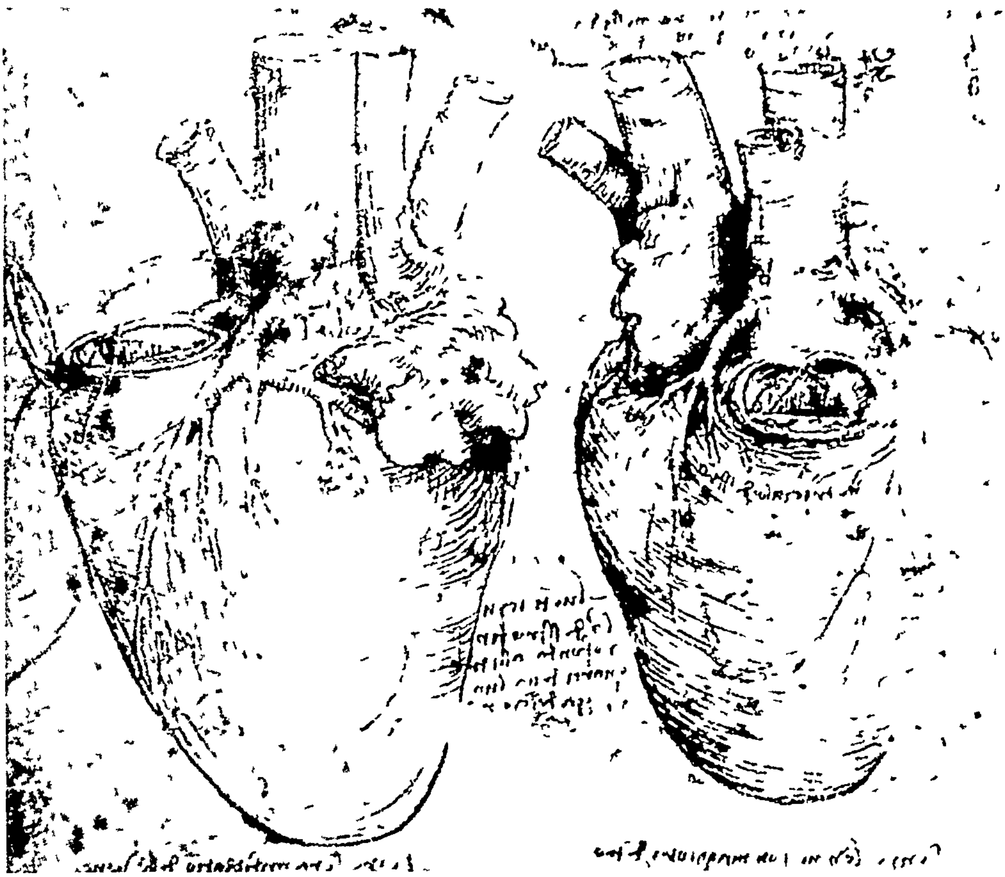
CARDIAC CLASSICS



Anatomic drawings of the heart by Leonardo da Vinci (1512)

(Courtesy Carnegie Institution of Washington)

CARDIAC CLASSICS

A Collection of Classic Works on the Heart and Circulation with Comprehensive Biographic Accounts of the Authors

Fifty-Two Contributions by Fifty-One Authors

BY

FREDRICK A. WILLIUS, M.D., M.S. in Med.

Chief, Section of Cardiology, The Mayo Clinic, Professor of Medicine, The Mayo Foundation for Medical Education and Research, The Graduate School, The University of Minnesota

AND

THOMAS E. KEYS, A.B., M.A.

Reference Librarian, The Mayo Clinic, Formerly Carnegie Fellow, The Graduate Library School, The University of Chicago

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If you do remember,
I send it through the rivers of your blood,
Even to the court, the heart, to the seat o' the brain
And through the cranks and offices of man,
The strongest nerves and small inferior veins
From me receive that natural competency
Whereby they live

—William Shakespeare Act 1, Scene 1, *Coriolanus*

FOREWORD

The era of specialization which has characterized medical education and practice in the past few years has greatly extended our knowledge of all fields of medicine. The advances represented in changing conceptions of disease, new methods of treatment, and radical revision of practice have introduced into medical education an important problem, that of correlation of accumulated facts and theories. An understanding of this correlation is difficult to attain but is equally essential to the medical student, the general practitioner, the specialist, and particularly the teacher. While it is usually true that advances in any field of endeavor have been accomplished through specialization, it is also true that the principles upon which practice should be based have been, and probably will continue to be, set up by those who possess an adequate knowledge of general medicine and can, therefore, correlate this with that acquired through intensive investigation of any special field. In the further development of specialization it is essential that education should focus on the fact that the broader the knowledge of general medicine, the more likely will investigations in any special field prove to be of permanent value.

Medical literature today represents these advances, and voluminous as the literature is, it has the virtue of reiteration, which is essential in education. In the appraisal of medical literature, students and practitioners are likely to overlook the writings of those who established the fundamentals upon which subsequent progress has been made. These epoch-making contributions are also evidence of the fact that in the study of disease thorough and accurate observation is the first requirement and supersedes other methods. In the diseases of the heart and circulation this fact is particularly true and in no field of medicine is sound clinical sense so important. The morbidity and mortality of heart disease is a challenge to the medical profession which is already being met, for recent statistics in this country show a decrease in this mortality rate. It is of particular importance, therefore, that any information which will aid the profession to understand better these diseases should be widely disseminated.

It is, therefore, appropriate that Dr. Willius and Mr. Keys have selected a representative group of classics pertaining to the heart and circulation and have added to the interest of these by accounts of the lives of the authors whose works are included and by a comprehensive correlation of the influence of these classics on the development of cardiology.

DONALD C. BALFOUR

Rochester, Minn.

PREFACE

The preparation of this volume of classics on the heart and circulation was occasioned by several motivating influences Our personal appreciation of the older medical writings was the first influence to encourage us to undertake this project After the expenditure of considerable time and effort, we came to the realization that the inaccessibility of numerous rare works undoubtedly deterred many physicians, as well as medical students, from availing themselves of these treasures of the past Furthermore, the accelerated tempo of modern times and the voluminous current medical literature allow many but little time for the culture of yesteryear However, the importance of these older writings remains unchallenged and many of them contain such accurate descriptions based on masterful observation that they endure as integral parts of the modern concepts of our day

One cannot read and contemplate the classics without being aroused by a desire to express apologies to the old masters, for there are many moderns who have written certain lines, believed to be original, only to find that the same observations and thoughts were expressed many years before It is reasonable to conclude that the classics of medical antiquity form the basis of modern medicine and that the physician of today relinquishes many cultural advantages when he avoids acquaintance with his distinguished predecessors It is our hope that this volume will reward the reader with the same measure of profit and pleasure that we have derived from the wisdom of these great masters

Dr Robert Watt of Glasgow more than 125 years ago wrote "The reading of the student is too often confined to systems and to compilations which are generally the work of men of no experience or of men writing under the influence of preconceived opinions To obtain correct views of medicine, it is necessary to have recourse to original authors, to such as write from actual observation, who have seen and treated the diseases they describe"

We do not presume that we have incorporated all the cardiac classics in the present volume, but we have selected from them those which have been of special interest to us and which, we believe, have contributed in a large measure to the development and progress of present-day cardiology We have selected contributions that deal with the anatomy and physiology of the heart and circulation, descriptions of disease, pathologic and therapeutic contributions, methods of diagnosis, and the like

We have attempted faithfully to reproduce the original writings, and where translations from other languages have been undertaken, it is our sincere hope that we have accurately expressed the author's meaning. With few exceptions we have reproduced the writings in their entirety, remembering our own feeling of keen disappointment in other years in reading a medical masterpiece that had been radically abridged solely by the personal discretion of the compiler. There are, however, instances in which the unusual length of a classic or tedious repetition made certain deletions advisable. It is our belief that the rather complete reproduction of these works is of much greater value and permits the reader more fully to appreciate and understand the personality and the philosophy of the author, than might be true had deletion been practiced.

We have presented the various classics in their chronologic order and have prefaced each with a biographic account of the author. Chronologic sequence has been carried out with the purpose of permitting the progress of cardiology to be recorded and compared with contemporary historic events. Whenever possible, note is made of discrepancies between the date of discovery and the date of publication of the contributions included. The order of their inclusion in this volume has been formulated according to date of publication; discrepancies are discussed either in the biographic accounts or in footnotes. Certain disagreement concerning dates occurs among various historians, and in so far as possible we have been guided by the date given by Garrison.

The first classic included in this volume is the epoch-making contribution of William Harvey, published in 1628. We have deliberately avoided earlier writings, being convinced that Harvey's work is in reality the fundamental contribution on which the modern concepts of the anatomy and physiology of the heart and circulation are based. Genuine progress in the field of cardiology first became evident when the views of Harvey became generally accepted. Our final classic is Dr. J. B. Herrick's account of coronary thrombosis which appeared in 1912.

We wish to acknowledge with gratitude the splendid resources of the Library of The Mayo Clinic and to express our appreciation for the splendid cooperation of its staff of librarians. We are deeply appreciative of the assistance rendered us by Dr. Maurice Walsh of the Mayo Clinic and Dr. Erich Hausner, formerly of the Mayo Foundation, in translations of works from the French and Latin. Our appreciation also is extended to Mr. James Eckman, our editor, of the Division of Publications of The Mayo Clinic, whose encouragement and suggestions have been of great help to us, and also many others whose technical assistance has been indispensable.

FREDRICK A. WILLIUS
THOMAS E. KEYS

Rochester, Minn.

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CARDIAC CLASSICS

THE INFLUENCE OF CERTAIN CARDIAC CLASSICS ON THE DEVELOPMENT OF MODERN CARDIOLOGY

IN THE history of medicine are recorded the heroic efforts of man over the unknown, the conflicts against ignorance, superstition, and prejudice, relentless self-sacrifice in the search for truth, indomitable courage in the face of failure and disappointment, all a part of the marvelous yet uncompleted pageant of present-day medicine In the chronologic presentation of these classics on the heart and circulation it is possible to trace the development of cardiology to its present position It is probable that the advancement of knowledge in the century to follow the present one will be greater and more dramatic than that of the three preceding centuries comprising the scope of this volume, but if this prediction materializes, it will be only because our illustrious predecessors have built their structure well and wisely

There is no doubt that William Harvey's epoch-making contribution, published in 1628, on the anatomy and the physiology of the heart and circulation laid the foundation for subsequent discoveries and advancements in this field Earlier observations are uncertain, isolated, and lack co-ordinated continuity, *some are erroneous and speculative, products of* the ages of scientific ignorance, religous prejudice, and superstition We do not mean to deny the fact that certain earlier observations were noteworthy, but their sporadic occurrence in the space of time hardly permits their inclusion in this consideration Even Harvey's views were bitterly contested by many of his contemporaries, who found it simpler to disagree on the premise of erroneous doctrines accepted as fact, rather than to open their minds to the comprehension of new data demonstrated by dissection and experiment Harvey proved the circulation of the blood and the manner in which it was accomplished, he also predicted the existence of the capillary circulation, which he referred to as "pores" He preceded the era of the microscope, and the use of his magnifying glass was of course inadequate for the demonstration of this minute anastomosis However, through remarkably clear reasoning he realized that some manner of communication between the terminal arterial and venous tributaries must exist in order to correlate the various observations which were evident from his thorough and painstaking investigations

In 1640, Pierre Gassendi recorded the demonstration of the existence of the foramen ovale in the adult heart Gassendi witnessed this demonstration, which was made by a surgeon named Payanus in Aix during the dissection of a human body This early settlement of a controversial

issue of that era is of unusual interest and reveals the meticulous care exercised by some of the older anatomists in carrying out their dissections

With the advent of and improvement in the microscope, the opportunity of demonstrating the capillary circulation, and thereby verifying Harvey's prediction, was accorded Marcello Malpighi, who vividly described these minute vascular structures in the lung and mesentery of the frog These observations were recorded in 1661, in his classic letters to Borelli, professor of science at Pisa, his friend and erstwhile idol

Three years later, in 1664, Niels Stensen revealed the true muscular nature of the heart This was a most important observation, because subsequent studies on the activity of the heart found confirmation in this fundamental demonstration

Thus, the teaching of correct basic anatomic and physiologic principles, though limited in scope, was the pioneer influence for the better understanding and recognition of departures from normal in the fields of physiology, pathology, and clinical medicine

In 1705, William Cowper clearly described disease of the aortic valve with aortic insufficiency This was accomplished through keen observation by correlating the symptoms of the patient and alterations detected in the pulse with the abnormal findings in the heart as revealed by postmortem examination It must be recalled that this observation antedated the discovery of auscultation by more than a century

Antony van Leeuwenhoek, in 1708, published his conception of "that motion which we call the pulse" Although his observations in this study were inaccurate and his conclusions erroneous, his noteworthy contributions in microscopy place him deservedly among science's immortals, for his mechanical ingenuity and inordinate patience enabled many brilliant successors to contribute generously to the advancement of cardiology

A quarter of a century later (1733) Stephen Hales, a scientifically minded clergyman, published the results of his rather unique experiments on blood pressure and blood velocity This was the first outstanding contribution on the physiology of the cardiovascular system following the exposition of Harvey These observations were made possible by the correct teaching of basic anatomic principles and yet little or nothing was known regarding hemodynamics These experiments, conducted on living animals, were the first in which an attempt at actual measurement of blood pressure and blood velocity in various parts of the circulatory system was undertaken They revealed certain fundamental facts upon which the modern concepts of today are largely dependent

In 1749, Jean Baptiste De Sénac made a most extraordinary observation which without a doubt is the very beginning of the relatively modern quinidine therapy of cardiac arrhythmia Although more than a century and a half elapsed before the medical world became cognizant of the

value of quinidine, De Sénac clearly described the beneficial effects of quinine in "rebellious palpitation."

Albrecht von Haller, in 1755, described calcification of the heart and pericardium in a very clear and vivid manner. This observation was based on a study of post-mortem material.

In 1761, the interesting and important observations of John Baptist Morgagni were published. They appeared in the form of letters collected in five books under the title of *The Seats and Causes of Diseases, etc.* (trans.). This work comprises the descriptions of a wide variety of diseases, with post-mortem observations. The remarkably accurate and interesting manner of presentation of Morgagni prompted us to reprint his observations on mitral stenosis, heart block, calcareous stenosis of the aortic valve with insufficiency, coronary sclerosis, and aneurysm of the aorta. Studies of this character were of great importance in the development of medicine because they represented the correlation of clinical symptoms and signs with dissection after death. Although the concepts of pathology were still ill-defined, observations of changes in tissue were recorded even though their nature and significance were not always understood. Such records served to stimulate others to pursue similar investigations with a determined curiosity.

Also in 1761, Leopold Auenbrugger published the results of his investigations dealing with a new diagnostic method, percussion of the thorax. This work preceded the introduction of auscultation by fifty-eight years. He introduced the Preface of his work with the following words, "I here present the Reader with a new sign which I have discovered for detecting diseases of the chest. This consists in the Percussion of the human thorax, whereby, according to the character of the peculiar sound thence elicited, an opinion is formed of the internal states of that cavity" (trans.). The tremendous influence of this discovery on the science and art of physical diagnosis requires no special comment. It enabled the physician to use an additional method of clinical investigation, since his methods were still limited to his own senses of perception.

In 1772, William Heberden's classic description of angina pectoris was first published, and again it appeared in 1802 in his *Commentaries on the History and Cure of Diseases*. Heberden, an outstanding scholar of his day, was possessed of the art of clear and accurate description, so that his vivid portrayal of the symptoms of angina pectoris, recorded in remarkable clarity, stands unchallenged today and is without a doubt one of the most cherished and brilliant masterpieces of the past.

William Withering, in 1785, published the results of years of study and observation and gave to the world a drug of inestimable and enduring value, digitalis. He was a botanist of wide experience, in addition to being a much respected physician. His concise comments on the use and actions of the foxglove *(Digitalis purpurea)* in cardiac dropsy, his remarkable

understanding of the indications and contraindications of the drug, his admonitions regarding its indiscriminate employment, and his warnings relative to overdosage endure today as the very basis of digitalis therapy Withering was one of science's most humble servants, as the introductory sentence of his Preface testifies "After being frequently urged to write upon this subject, and as often declining to do it, from apprehension of my own inability, I am at length compelled to take up the pen, however unqualified I may still feel myself for the task" His dissertation becomes more interesting and remarkable when it is recalled that the era in which he lived saw therapeutics in the zenith of empiricism Drugs of unknown and uncertain action were administered by the method of trial and error, standardization of preparations was unknown or uncertain, and therapeutics to a large extent became a tradition perpetuated by word of mouth

In 1788, Matthew Baillie picturesquely described the findings after death in a case of congenital dextrocardia with complete situs transversus In this publication he philosophized in a very interesting and understanding manner on the origin of the condition Baillie is particularly noted for his interest in pathology, and he was probably the first physician to emphasize morbid anatomy as a definitive branch of medical science He investigated the morbid changes in various organs and attempted to correlate them in the individual case His studies unquestionably wrought an important influence in the field of pathology and in no small measure contributed to the groundwork of that science

John Hunter, the celebrated English surgeon, rather unwittingly contributed to the progress of cardiology through the personal records of his own illness He suffered severe, recurrent attacks of angina pectoris, and his observations and descriptions of the symptoms and signs of the disorder were the first recorded by a physician suffering from this disease The intimate aspects of his illness are extremely interesting, as are also his various and sundry therapeutic attempts at relief in an era in which therapeutic effort was, to say the least, extremely discouraging This account was published by his brother-in-law, Everard Home, in 1794, three years after Hunter's death Post-mortem examination of Hunter revealed marked sclerosis of the coronary arteries which was vividly described in the following words "The coronary arteries had their branches which ramify through the substance of the heart in the state of bony tubes, which were with difficulty divided by the knife, and their transverse sections did not collapse, but remained open" This is one of the early accounts demonstrating the coexistence of angina pectoris and coronary sclerosis

In 1806, Jean Nicolas Corvisart published his treatise on diseases of the heart This was a comprehensive consideration of the diseases of the heart and great vessels according to the knowledge of that era He

emphasized percussion as an important diagnostic method, and his influence undoubtedly had great influence in its survival, for Auenbrugger's teachings had not been enthusiastically received by his contemporaries. Laennec, one of Corvisart's illustrious students, was greatly stimulated by his able teacher, as evidenced by the frequent citation of Corvisart in Laennec's treatise on auscultation.

William Charles Wells, in 1812, published one of the earliest clinical accounts of the cardiac participation in rheumatic fever, which he designated as rheumatism of the heart. His observations preceded the discovery of auscultation and his observations comprised the record of symptoms referable to the heart and alterations occurring in the pulse, notably tachycardia and irregularity. In several instances post-mortem examination confirmed Wells's clinical suspicions. The recognition of rheumatic fever as a causative factor in heart disease was to become an important contribution in the field of etiology.

Clinical observations were recorded with greater frequency as the nineteenth century progressed, and one of the striking developments was the evident desire to correlate symptoms and signs of disease with post-mortem observations. A typical instance of keen observation was that of John Cheyne, who, in 1818, described an unusual form of periodic breathing in an instance of fatty heart, which was again described in 1846 by William Stokes, and ultimately became known as "Cheyne-Stokes respiration." In both of these accounts of the disorder, the descriptions are vividly clear and impressive, and testify to the art of careful observation and expression.

In 1819 a new and very fruitful method of clinical examination was introduced in René T. H. Laennec's epoch-making contribution of auscultation. With the acceptance and refinement of this method, progress in the diagnosis of diseases of the heart advanced in great strides. This discovery was, in a large measure, directly responsible for the great advances in cardiology that were destined to occur in Laennec's century. In fairly rapid succession, many important observations and discoveries ensued, of which we are able to reproduce only certain outstanding classics.

Caleb Hillier Parry, in 1768, was the first to recognize exophthalmic goiter and its cardiovascular phenomena. This work, however, was not published until 1825.

Two years later (1827) Robert Adams presented his classic description of heart block, which was again described by William Stokes in 1854, and the cerebral phenomena at times present in this disorder later became known as the "Adams-Stokes syndrome."

In 1831, James Hope published a remarkably complete treatise on diseases of the heart and great vessels. Numerous interesting and graphic descriptions are contained in this work. Of unusual interest are his dis-

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*We again wish to remind the reader that the chronologic sequence of presentation in this volume is governed by the year of publication and not necessarily by the year in which the observation or work was carried out.—F. A. W. 1940

cussions of cardiac asthma, stenosis of the pulmonary valves, and cardiac neurosis. Of particular importance is Hope's discussion of cardiac asthma, because this syndrome was then only rarely identified as being of cardiac origin.

Sir Dominic John Corrigan, in 1832, published his classic description of the pulse in aortic insufficiency. This observation was important from more than the standpoint of diagnosis, for it stimulated interest and thought on the vascular mechanics of this valvular lesion. Here again is portrayed accurate and masterful description of keen observation.

In 1835, Jean-Baptiste Bouillaud, an eminent physician of his era, among other important contributions described the pathologic aspects of endocarditis according to the knowledge of his time and undoubtedly stimulated others to inquire further into the endocarditides.

Six years later, William Senhouse Kirkes described and discussed emboli resulting from intracardiac coagula and corrected the existing tenets ascribing the remote lesions to local disease (so-called capillary phlebitis). This work was especially important in supporting the theory of emboli. Minute and gross visceral infarction is described in a clear and understanding manner.

Additional observations regarding the signs of already recognized diseases began to appear, and in 1861 Paul Louis Duroziez described the auscultatory findings audible in certain peripheral arteries, notably the femoral arteries, in the presence of aortic insufficiency. He referred to this phenomenon as the "double intermittent murmur over the femoral (crural) arteries" (trans.), which later was to be known as "Duroziez' sign."

In this era of medicine, the teaching of clinical cardiology largely centered around murmurs, and much emphasis was placed on the description of their character, slight variations in timbre and intensity, timing and transmission. This period of the history of cardiology witnessed many significant advances and stimulated physicians to search for new and additional diagnostic methods to penetrate still further the secrets of the normal, as well as of the diseased, heart. It was in 1862 that Austin Flint, an American physician, published his important contribution, "On Cardiac Murmurs," which appeared in the *American Journal of the Medical Sciences*. This paper deals comprehensively with murmurs in general and contains the description of the murmur, later to be known as the "Austin Flint murmur."

In 1867, Pierre Carl E. Potain called attention to the importance and significance of another peripheral phenomenon of heart disease in his observations on the pulsations of the jugular veins. Evidence more or less remote from the heart was gradually being accumulated and correlated, helping to lay the foundation for the innumerable signs that now constitute the diagnostic armamentarium of the present-day physician.

In the same year, Sir Thomas Lauder Brunton enriched the world with the important contribution relative to the ameliorating effects of amyl nitrite in the anginal syndrome. Preceding this discovery, sufferers from angina pectoris had received little or no relief from the therapeutic agents available to them. In this study the vasodilating action of amyl nitrite, first suspected by Dr. B. W. Richardson, was confirmed. The drug was discovered by Balard, but Brunton was the first to suggest its use on a practical therapeutic basis. Studies of this character were extremely important in the attack on the existing therapeutic empiricism of that era.

A year later (1868) Heinrich Irenaeus Quincke clearly described the capillary and venous pulse. Quincke was another keen observer who called attention to more remote signs of the impaired heart. These observations were significant and continue to be of great practical importance today.

In 1870, Sir Samuel Wilks very clearly described the disease later to be known as "bacterial endocarditis," under the title "capillary embolism or arterial pyaemia." This contribution was important in that it called attention to the necessity for separating old valvular defects (healed endocarditis) from associated or superimposed vegetative lesions resulting in the dissemination of emboli.

Ludwig Traube, two years later (1872), described a significant disturbance in the pulse which he termed "pulsus alternans." He clearly distinguished it from the simulating condition, pulsus bigeminus. Traube's contribution has endured as a very valuable sign and today continues to guide the clinician in his appraisal of the course of the failing heart.

In 1876, Sir William Richard Gowers graphically described certain changes found in the retinal vessels in the presence of arterial hypertension. This demonstration was destined to be of great importance, as testified to by the extensive development of retinoscopy in recent years. He fully appreciated the fact that the retinal arteries, visible by special means, afforded the physician an opportunity of actually observing vessels during the life of the patient and of comparing the retinas of normal individuals with those of patients afflicted with cardiovascular-renal disease. At the time that Gowers conducted these studies, the concept of primary renal damage occupied a very prominent position, and the concept of generalized vascular disease in relationship to hypertension had not yet been clearly conceived.

A year later (1877), Julius Friedrich Cohnheim in his chapter on thrombosis and embolism of his work on the *Pathology of the Circulation* (trans.) described paradoxical embolism. This work called attention to a new significance attending otherwise innocuous imperfections of the septa of the heart and demonstrated the manner in which, in the presence of these imperfections, thrombi arising in the venous system can be transported to the periphery of the arterial system.

In 1879, Henri Roger described the uncomplicated congenital defect of the interventricular septum and masterfully described the prolonged murmur, extending through both systole and diastole, pathognomonic of this defect. The murmur is still frequently known as the "Roger murmur."

In the same year, William Murrell, undoubtedly influenced by the work of his worthy predecessor, Sir Thomas Lauder Brunton, published his studies on the effects of nitroglycerin in angina pectoris. In this investigation, conducted during a controversial period because of a lack of standardization of drugs, he pursued his studies in a determined manner and compared the effects of nitroglycerin with those of amyl nitrite. This contribution made available another valuable drug for the mitigation of the anginal seizure. It also stressed the importance of standardization of drugs.

Six years later (1885) Pierre Carl Potain made another important contribution to cardiology. He published his studies on gallop rhythm and presented his views on the genesis and significance of this condition.

Augustus D. Waller, in 1887, after extensive physiologic studies on the action currents of the heart by means of the capillary electrometer, published his work on a method of leading the currents from the surface of the body by means of paired electrodes. Previous work in this field had necessitated direct contact with the heart. His studies clearly paved the way for clinical electrocardiography, an almost indispensable method of modern cardiology.

In the same year, John Alexander MacWilliam recorded the results of his experiments dealing with direct faradization of the heart, and produced profound alterations in rate and rhythm. He clearly described the abnormal rhythms now known as "auricular fibrillation," "auricular flutter," "ventricular tachycardia" and "ventricular fibrillation." His work, moreover, was a steppingstone to clinical electrocardiography, particularly from the standpoint of the ultimate correlation of graphs revealing abnormal waves with actual visualization of induced disturbances in the hearts of animals.

A year later (1888), Graham Steell described the pulmonary diastolic murmur, under the title of "The Murmur of High Pressure in the Pulmonary Artery." This was destined to be known as the "Graham Steell murmur," and is so designated today.

In the same year, A. Fallot described the interesting and unusual combination of congenital cardiac defects consisting of pulmonary stenosis, interventricular septal defect, dextroposition of the aorta, and hypertrophy of the right ventricle. This became known as the *tetralogy of Fallot* and is frequently referred to as the *maladie bleue*.

At this time, very little was known regarding the intrinsic physiology of the cardiac impulse and its dissemination through the substance of the

heart. It was in 1893 that Wilhelm His, Jr., accurately described the minute fasciculus that conducts the impulse from the auricles to the ventricles: the auriculoventricular bundle. This structure is still frequently designated as the "bundle of His." This contribution was of paramount importance in the clearer understanding of the intricate mechanism of cardiac conduction and inevitably led to further discoveries in this field.

In 1896, Francis Henry Williams of Boston reported his observations on the fluoroscopic examination of the heart and aorta. Although his studies were not the first recorded, they are extremely noteworthy and we have chosen to include them in this volume. Williams' observations were published a year following Roentgen's original observations and surely represent pioneer efforts in this remarkable branch of science.

The following year (1897), Sir William Henry Broadbent presented his classic description of adherent pericarditis and described the recession of the intercostal spaces as a sign of this disease, a sign which was to become known as "Broadbent's sign." Simulating findings have since been frequently misinterpreted, yet Broadbent emphatically discussed the limitations of interpretation.

In 1903, Willem Einthoven devised the string galvanometer, the original modern electrocardiograph. During his extensive experience with the capillary electrometer in the study of the action currents of the heart, he was aware of the inherent error existing in this method and sought to devise a method of registration wherein this error would be obviated. The world owes this humble scientist a tremendous debt of gratitude for his brilliant gift, for with the advent of electrocardiography, remarkable progress has been made, and many of the secrets of the heart have gradually become bared. Electrocardiography is today virtually an indispensable method in the thorough appraisal of the heart.

A year later (1904), during the time that the issue between the myogenic and neurogenic theories of heart contraction was controversial, Ludwig Aschoff presented his epoch-making work on rheumatic myocarditis. He described the characteristic lesion of rheumatic fever, which has come to be known as the "Aschoff nodule." This was a monumental work and did much to crystallize the present-day concepts of the pathology of this yet unsolved disease, and it established a histopathologic picture that was destined to exert a great influence in classifying examples of true myocarditis. Other myocardial abnormalities, later to be proved noninflammatory in nature, were before this universally shuffled into the rather vague category of "myocarditis."

It was not until 1907 that the sino-auricular node was demonstrated by the painstaking microscopic studies of Arthur Keith and Martin Flack. They also predicted the function of this node to be the "pacemaker" of the heart, that is, the point of origin of the cardiac impulse. This discovery proved to be of great importance in bridging certain gaps

in the more comprehensive understanding of the intrinsic anatomy and physiology of the heart.

Sir James Mackenzie contributed generously to the modern concepts of cardiology, but the identification and clarification of auricular fibrillation was probably one of his greatest achievements. The conclusive publication of this work appeared in 1908, although he described this form of arrhythmia in 1902, believing then that it represented "nodal rhythm." His clinical observations on the pulse and the differentiation and segregation of the various irregularities of the pulse were made possible by his adoption of the simultaneous recording of arterial and venous pulse waves by means of the polygraph. This mechanical method of registration was eventually supplanted by the electrocardiograph, in a great measure through the comprehensive studies of Mackenzie's brilliant student, Sir Thomas Lewis.

At this time another outstanding physician was molding the form of modern medicine, and his varied contributions are still the byword of present-day medical students. Sir William Osler's influence on modern medicine was profound. We have chosen to reproduce his article on chronic infectious endocarditis, published in 1909, wherein the description of the cutaneous nodules of subacute bacterial endocarditis is found. These pathognomonic lesions are widely known today as "Osler's nodes."

Our final contributor, one of the few masters still living, is our beloved American, Dr. James Bryan Herrick. His classic description of coronary thrombosis, appearing in 1912 in the *Journal of the American Medical Association,* clearly opened a new era in which the clinical recognition of this disease was to become the rule rather than the exception. Although this report was preceded by that of Dr. Adam Hammer of St. Louis and Vienna in 1878, Herrick's paper attracted much wider attention and therefore appears to have had a more profound influence on the modern concepts of the disease than that of Hammer.

It has been impossible to include all noteworthy contributions of the past in this volume of CARDIAC CLASSICS, and there will be some critics who will disapprove of certain inclusions while others will question certain exclusions. In the anticipation of such criticisms we can but state, simply, that we have conscientiously attempted to make this collection of classics comprehensive, and have selected from the great literature of the three centuries preceding our own those contributions which have particularly appealed to us and which, we believe, have profoundly influenced the progress of this branch of medical science. We have, as far as possible, avoided the inclusion of the works of living physicians, well realizing the possibilities for controversy which would have arisen if such a plan had been extensively adopted. There are, of course, many notable contributions by our living contemporaries, who, however, must remain patient until some future compilers honor them in a manner similar to that adumbrated by the present collection.

1628

WILLIAM HARVEY

ON THE MOTION OF THE HEART AND BLOOD IN ANIMALS

WILLIAM HARVEY

Painting by unknown contemporary

(Courtesy Ciba Symposia)

WILLIAM HARVEY

(1578-1657)

"Cor, Imperator, Rex"

"I was almost tempted to think with Fracastorius that the motion of the heart was only to be comprehended by God"

—William Harvey, Chapter I, *De motu cordis*

WILLIAM HARVEY, born in 1578 as the eldest son of Thomas Harvey of Folkestone, Kent, had his preliminary education at the Canterbury Grammar School and was admitted as a student of Caius' College, Cambridge, in 1593 Dr Caius, the founder and long the master according to Power (p 13), in addition to his knowledge of Greek, introduced the study of practical anatomy into England Through his influence, his college was allowed to use the bodies of two criminals annually for the purposes of dissection It is not definitely known whether or not Harvey was permitted to watch or to participate in dissections, but it is known that he was graduated from this school, Bachelor of Arts, in 1597 His collegiate education probably was a general one and included a sound knowledge of Greek and Latin plus an acquaintanceship with dialectics and with physics

In choosing Padua for the study of medicine, Harvey was no doubt drawn by the renown of its medical school, made famous by the great Vesalius and by the work of the successor of Vesalius, Hieronymus Fabricius Harvey supposedly entered Padua in 1598, but there is no record of his being there before the year 1600

The theater in which Fabricius lectured still exists It is now an ancient structure containing seats which rise perpendicularly, one above the other. But when Harvey was at Padua the theater was new, and the government had placed an inscription over the entrance to commemorate the genius of Fabricius Fabricius must have been a source of great inspiration to Harvey and it was under him that the young student became an expert in anatomy Harvey makes reference in "De motu cordis" to his former teacher as "the celebrated Hieronymus Fabricius of Aquapendente, a most skilful anatomist, and venerable old man" While Harvey was receiving his medical training at Padua, Fabricius was perfecting his knowledge concerning the valves of the veins Sylvius of Louvilly (Jacques DuBois, 1478-1555), the teacher of Vesalius at Paris, had known and described the valves at an early date But Fabricius rediscovered them in 1574 (Power, p 25) Fabricius, as Harvey so skillfully points out, did not rightly understand the function of the valves Fabricius thought their purpose was to prevent overdistention of the vessels when blood flowed from the larger into the smaller veins, but that they were not needed in the arteries because the blood was always in a state of ebb and flow[1] Harvey pointed out that the true function of the valves was to prevent venous reflux and, therefore, this prevention of contrary motion was a great factor in the circulation of the blood

After a five-year stay at Padua, Harvey received his diploma as Doctor of Physics (1602), with the right to practice and teach arts and medicine in every land and seat of learning Apparently, Harvey had made a great impression on his superiors, for his diploma further stated, "He had conducted himself so wonderfully well in the

WILLIAM HARVEY EXPOUNDING HIS CONCEPTION OF THE CIRCULATION OF THE BLOOD TO KING CHARLES I OF ENGLAND

Painting by Robert Hannah

(Courtesy Ciba Symposia)

examination, and had shown such skill, memory, and learning that he had far surpassed even the great hopes which his examiners had formed of him'' (Quoted by Power, pp 26-27)

On his return to England in the same year, Harvey also received the degree of Doctor of Medicine from the University of Cambridge Two years later (1604), Harvey settled in London, married the daughter of a physician (Lancelot Browne, physician to Queen Elizabeth and to King James I) by whom, it is to be regretted, he had no children He then entered the practice of his profession and was elected a fellow of the College of Physicians in 1607 In 1609 he was duly appointed physician to St Bartholomew's Hospital

In 1615 Harvey was appointed to the office of Lumleian lecturer, a highly esteemed position under the sponsorship of the College of Physicians He continued in this capacity until 1656, when he resigned his post Harvey's lectures (on anatomy and surgery as qualified by the Lumleian Trust) were first delivered from April 16 to 18, 1616. On April 23 of that same year, Shakespeare died at Stratford-on-Avon. Garrison (p 249) and others have pointed out the resemblance of Harvey's finely formed head to that of the world's greatest dramatist And it may be possible that Harvey was influenced somewhat by his great contemporary

The manuscript notes of his first course of lectures are now the sacred property of the British Museum The second portion of his notes (as described by Power, pp 64-66) contains an account of the thorax and its contents After a full discussion of the situation and functions of the various abdominal viscera, Harvey next considered the thorax, and his remarkable words contain his first written description of his memorable discovery These words are initialed to show that Harvey believed the idea was original

''It is plain from the structure of the heart that the blood is passed continuously through the lungs to the aorta as by the two clacks of a water bellows to raise water

''It is shown by the application of a ligature that the passage of the blood is from the arteries into the veins

''Whence it follows that the movement of the blood is constantly in a circle, and is brought about by the beat of the heart It is a question, therefore, whether this is for the sake of nourishment or rather for the preservation of the blood and the limbs by the communication of the heat, the blood cooled by warming the limbs being in turn warmed by the heart''

Therefore, it is apparent that Harvey knew of the circulation, at least, by the year 1616, at the age of thirty-seven, and twelve years before the publication of ''De motu cordis''

In 1618 Harvey was appointed Physician Extraordinary to James I and, on the death of that monarch, his son, Charles I, appointed Harvey a physician-in-ordinary Besides being physician to the King's household, Harvey was physician to several distinguished noble families Included among his patients was Francis Bacon (1561-1626), whose great genius did not impress the mind of Harvey, who said of one of Bacon's works, ''He writes philosophy like a Lord Chancellor''

The year 1628 marks the highest point in the career of William Harvey From Frankfort-on-the-Main came his matured account in Latin of the circulation of the blood The Italians claimed Andrea Cesalpino[1] (1524-1603), professor of medicine

[1]Erasistratus (circa 310-250 B C) of Keos, the first experimental physiologist, described the aortic and pulmonary valves, the chordae tendineae of the heart and had the idea of the capillary system He also conceived that the heart was a pump and expressed the first theory of the circulation (See Finlayson J Hierophilus and Erasistratus Glasgow M J 39 321-352, 1893) Others in addition to Cesalpino who contributed more or less meritorious theoretical accounts of the circulatory system included Ibn an- Nafis, of Egypt and Syria (c 1288-1289), Servetus (1509-1553), the Spaniard, and Realdo Colombo (1516-1559), the successor of Vesalius at Padua

at Pisa, as having discovered the circulation during the period from 1571-1593 prior to Harvey (1616) Cesalpino described the lesser circulation, but his observations did not lead him to a clear recognition of the greater circulation Cesalpino is deserving of credit, however, and it is possible that Harvey knew of his work while he was a student at Padua In his first chapter of "De motu cordis," Harvey suggests that he sought to discover the motions and uses of the heart "from actual inspection and not from the writings of others" For this purpose he resorted to vivisection, ligation and perfusion He experimented on several living animals whose hearts were observable with the naked eye, and also on some smaller animals whose hearts he could observe with the aid of a magnifying glass As an anatomist, and an outstanding one, he was further able to confirm much of his proof of the circulation on the basis of his many dissections of human bodies

The mainstay of Harvey's argument—that the actual quantity of blood as measured made it physically impossible for the blood to do other than return to the heart by the venous route—not only gave him definite proof of the circulation but also, as Garrison (p 247) has so well stated, insured that his computation was the first idea of measurement in any biologic investigation This quantitative demonstration gave impetus to the rise of physiology

After the publication of "De motu cordis," there can be no doubt that Harvey's prestige as a physician suffered somewhat He was severely attacked by the pedantic thinkers of the time But this disturbance was more than counterbalanced by the acceptance during his lifetime of his most important contribution

Harvey, at an early date (1613), had been elected to the office of censor in the College of Physicians He was reappointed to this office in 1625 and again in 1629 In 1628 he was appointed Treasurer of the "College," to which office he was reappointed in 1629 In the same year he received the commands of the King to accompany the Duke of Lennox, who was sent to travel abroad He continued to travel with the Duke until the winter of 1631-1632, at which time he returned to England In 1632 he drew up a set of rules for the new library of the College of Physicians Early in 1633, Harvey again received the commands of the King, this time to attend Charles I on his journey to Scotland During this trip he wrote his treatise on the "Bass Rock"

In 1634, the story of the Lancashire witches ran rampant through England As was the case in the Salem witchcraft episode in New England in 1692, the accusations were the result of a child's perjury Dr Harvey was called upon to examine the bodies of some of the arrested "witches" and some of those permitted to live Owing to his testimony and that of others, four of the seven convicted "witches" were pardoned Harvey's attitude is all the more remarkable, considering that his contemporary in medicine, Sir Thomas Browne (1605-1682), supposedly a model of tolerance, affirmed that he believed in witchcraft (Smith, vol 1, p 445), and the great Carl Linnaeus (1707-1778), born 129 years later than Harvey, displayed in his "Nemesis Divina," written for his son, that he believed in supernatural punishments (Smith, vol 2, pp 540, 541)

Another interesting experience occurred to Harvey in 1635 In this year he was ordered by the King to perform necropsy on the body of Thomas Parr, who is said to have died at the ripe age of one hundred and fifty-two years and nine months The notes of this necropsy were not printed until 1669, when they were published in Bett's "On the Source and Quality of Blood" From these notes it appears that Harvey believed "Old Tom Parr" would have lived even longer had he remained in his native home, Shropshire, and not transported himself to London and altered his diet by living with a nobleman!

Harvey's friendship with the King resulted in his being suspected, and rightly, of being an enthusiastic Loyalist In the early part of the Civil War (1642), a mob

of citizen-soldiers entered Harvey's lodgings, stole his goods, and scattered his papers These papers included the records of a large number of dissections, his observations on the development of insects, and a series of notes on comparative anatomy

In 1645 Harvey was elected to the honorable position of warden of Merton College at the University of Oxford This was the school of which the famous John of Gaddesden (circa 1350), one of the earliest Englishmen to write a complete treatise on medicine,[2] had been a fellow Because of the tumult of the Cromwellian Civil War, Harvey held this position for only one year

The surrender of Oxford in 1645 marks the period of Harvey's severance from the Court and his gradual retirement from public life, occasioned in part by his being affected with gout, from which he suffered recurrent attacks During this time he was preparing his essay "De generatione animalium," which was published in 1651 In his investigation of the embryo, handicapped as he was by not having a microscope, he formed a wrong idea of fecundation He believed the fertilization of the ovum to be something "incorporeal—as iron touched by the magnet is endowed with its own powers" Garrison (p 248) suggests that the true importance of "De generatione" was "that it subverted the ancient concept that life is engendered out of corruption (or putrefaction) "

The College of Physicians was promised, by an anonymous donor, a library of books, a museum of numerous objects of curiosity, and a variety of surgical instruments Before the building was completed the name of the generous benefactor became known, and the College responded in 1652 by creating a statue of Harvey

But the College still felt indebted to Harvey and chose him as its president in 1654 He, however, did not accept this honor, recognizing the influences of the infirmities in his health The attacks of gout continued, and he died on June 3, 1657, from a cerebral hemorrhage He was buried in the family vault at Hempstead in Essex

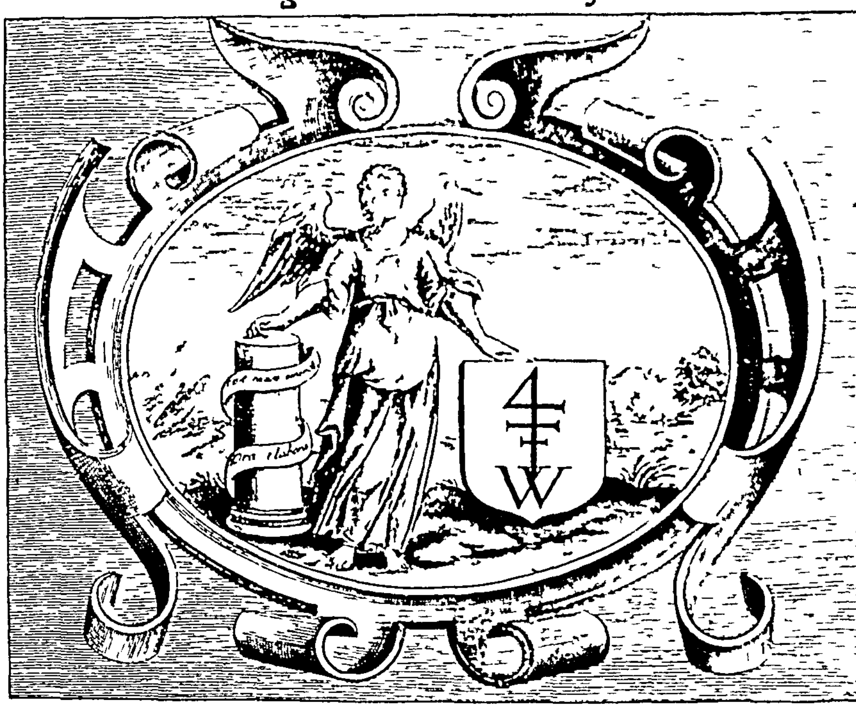
Abraham Cowley (1618-1667) wrote of Harvey

> "Harvey sought for Truth, in Truth's own book,
> The creatures, which by God himself was writ,
> And wisely thought 'twas fit,
> Not to read comments only upon it,
> But on th' original itself to look "

[2] *Rosa Anglica,* printed in 1492

EXERCITATIO ANATOMICA DE MOTV CORDIS ET SANGVINIS IN ANIMALIBVS,

GVILIELMI HARVEI ANGLI,
Medici Regii, & Professoris Anatomiæ in Collegio Medicorum Londinensi.



FRANCOFVRTI,
Sumptibus GVILIELMI FITZERI.
ANNO M DC. XXVIII.

AN ANATOMICAL DISQUISITION ON THE MOTION OF THE HEART AND BLOOD IN ANIMALS*

By

WILLIAM HARVEY, M.D.

INTRODUCTION

AS WE are about to discuss the motion, action, and use of the heart and arteries, it is imperative on us first to state what has been thought of these things by others in their writings, and what has been held by the vulgar and by tradition, in order that what is true may be confirmed, and what is false set right by dissection, multiplied experience, and accurate observation

Almost all anatomists, physicians, and philosophers, up to the present time, have supposed with Galen, that the object of the pulse was the same as that of respiration, and only differed in one particular, this being conceived to depend on the animal, the respiration on the vital faculty, the two, in all other respects, whether with reference to purpose or to motion, comporting themselves alike Whence it is affirmed, as by Hieronymus Fabricius of Aquapendente, in his book on "Respiration," which has lately appeared, that as the pulsation of the heart and arteries does not suffice for the ventilation and refrigeration of the blood, therefore were the lungs fashioned to surround the heart From this it appears, that whatever has hitherto been said upon the systole and diastole, on the motion of the heart and arteries, has been said with especial reference to the lungs

But as the structure and movements of the heart differ from those of the lungs, and the motions of the arteries from those of the chest, so seems it likely that other ends and offices will thence arise, and that the pulsations and uses of the heart, likewise of the arteries, will differ in many respects from the heavings and uses of the chest and lungs For did the arterial pulse and the respiration serve the same ends, did the arteries in their diastole take air into their cavities, as commonly stated, and in their systole emit fuliginous vapours by the same pores of the flesh and skin, and further, did they, in the time intermediate between the diastole and the systole, contain air, and at all times either air, or spirits, or fuliginous vapours, what should then be said to Galen, who wrote a book on purpose to show that by nature the arteries contained blood, and nothing

Exercitatio anatomica de motu cordis et sanguinis in animalibus, London 1628 Translated by Robert Willis, Barnes Surrey, England, 1847

but blood, neither spirits nor air, consequently, as may be readily gathered from the experiments and reasonings contained in the same book? Now if the arteries are filled in the diastole with air then taken into them (a larger quantity of air penetrating when the pulse is large and full), it must come to pass, that if you plunge into a bath of water or of oil when the pulse is strong and full, it ought forthwith to become either smaller or much slower, since the circumambient bath will render it either difficult or impossible for the air to penetrate In like manner, as all the arteries, those that are deep-seated as well as those that are superficial, are dilated at the same instant, and with the same rapidity, how were it possible that air should penetrate to the deeper parts as freely and quickly through the skin, flesh, and other structures, as through the mere cuticle? And how should the arteries of the foetus draw air into their cavities through the abdomen of the mother and the body of the womb? And how should seals, whales, dolphins and other cetaceans, and fishes of every description, living in the depths of the sea, take in and emit air by the diastole and systole of their arteries through the infinite mass of waters? For to say that they absorb the air that is infixed in the water, and emit their fumes into this medium, were to utter something very like a mere figment And if the arteries in their systole expel fuliginous vapours from their cavities through the pores of the flesh and skin, why not the spirits, which are said to be contained in these vessels, at the same time, since spirits are much more subtile than fuliginous vapours or smoke? And further, if the arteries take in and cast out air in the systole and diastole, like the lungs in the process of respiration, wherefore do they not do the same thing when a wound is made in one of them, as is done in the operation of arteriotomy? When the windpipe is divided, it is sufficiently obvious that the air enters and returns through the wound by two opposite movements, but when an artery is divided, it is equally manifest that blood escapes in one continuous stream, and that no air either enters or issues If the pulsations of the arteries fan and refrigerate the several parts of the body as the lungs do the heart, how comes it, as is commonly said, that the arteries carry the vital blood into the different parts, abundantly charged with vital spirits, which cherish the heat of these parts, sustain them when asleep, and recruit them when exhausted? And how should it happen that, if you tie the arteries, immediately the parts not only become torpid and frigid, and look pale, but at length cease even to be nourished? This, according to Galen, is because they are deprived of the heat which flowed through all parts from the heart, as its source, whence it would appear that the arteries rather carry warmth to the parts than serve for any fanning or refrigeration Besides, how can the diastole (of the arteries) draw spirits from the heart to warm the body and its parts, and, from without, means of cooling or tempering them? Still further, although some affirm that the lungs, arteries, and heart have all the same offices, they yet maintain that the heart is the workshop of the spirits, and that the arteries

contain and transmit them, denying, however, in opposition to the opinion of Columbus, that the lungs can either make or contain spirits, and then they assert, with Galen, against Erasistratus, that it is blood, not spirits, which is contained in the arteries.

These various opinions are seen to be so incongruous and mutually subversive, that every one of them is not unjustly brought under suspicion. That it is blood and blood alone which is contained in the arteries is made manifest by the experiment of Galen, by arteriotomy, and by wounds, for from a single artery divided, as Galen himself affirms in more than one place, the whole of the blood may be withdrawn in the course of half an hour, or less. The experiment of Galen alluded to is this: "If you include a portion of an artery between two ligatures, and slit it open lengthways, you will find nothing but blood", and thus he proves that the arteries contain blood only. And we too may be permitted to proceed by a like train of reasoning: if we find the same blood in the arteries that we find in the veins, which we have tied in the same way, as I have myself repeatedly ascertained, both in the dead body and in living animals, we may fairly conclude that the arteries contain the same blood as the veins, and nothing but the same blood. Some, whilst they attempt to lessen the difficulty here, affirming that the blood is spiritous and arterious, virtually concede that the office of the arteries is to carry blood from the heart into the whole of the body, and that they are therefore filled with blood, for spiritous blood is not the less blood on that account. And then no one denies that the blood as such, even the portion of it which flows in the veins, is imbued with spirits. But if that portion which is contained in the arteries be richer in spirits, it is still to be believed that these spirits are inseparable from the blood, like those in the veins, that the blood and spirits constitute one body (like whey and butter in milk, or heat [and water] in hot water), with which the arteries are charged, and for the distribution of which from the heart they are provided, and that this body is nothing else than blood. But if this blood be said to be drawn from the heart into the arteries by the diastole of these vessels, it is then assumed that the arteries by their distension are filled with blood, and not with the ambient air, as heretofore, for if they be said also to become filled with air from the ambient atmosphere, how and when, I ask, can they receive blood from the heart? If it be answered: during the systole, I say, that seems impossible, the arteries would then have to fill whilst they contracted, in other words, to fill, and yet not become distended. But if it be said during the diastole, they would then, and for two opposite purposes, be receiving both blood and air, and heat and cold, which is improbable. Further, when it is affirmed that the diastole of the heart and arteries is simultaneous, and the systole of the two is also concurrent, there is another incongruity. For how can two bodies mutually connected, which are simultaneously distended, attract or draw anything from one another, or, being simultaneously contracted, receive anything from each other? And then

it seems impossible that one body can thus attract another body into itself, so as to become distended, seeing that to be distended is to be passive, unless, in the manner of a sponge, previously compressed by an external force, whilst it is returning to its natural state But it is difficult to conceive that there can be anything of this kind in the arteries The arteries dilate, because they are filled like bladders or leathern bottles, they are not filled because they expand like bellows This I think easy of demonstration, and indeed conceive that I have already proved it Nevertheless, in that book of Galen headed 'Quod Sanguis continetur in Arteriis,' he quotes an experiment to prove the contrary An artery having been exposed, is opened longitudinally, and a reed or other pervious tube, by which the blood is prevented from being lost, and the wound is closed, is inserted into the vessel through the opening "So long," he says, "as things are thus arranged, the whole artery will pulsate, but if you now throw a ligature about the vessel and tightly compress its tunics over the tube, you will no longer see the artery beating beyond the ligature" I have never performed this experiment of Galen's, nor do I think that it could very well be performed in the living body, on account of the profuse flow of blood that would take place from the vessel which was operated on, neither would the tube effectually close the wound in the vessel without a ligature, and I cannot doubt but that the blood would be found to flow out between the tube and the vessel Still Galen appears by this experiment to prove both that the pulsative faculty extends from the heart by the walls of the arteries, and that the arteries, whilst they dilate, are filled by that pulsific force, because they expand like bellows, and do not dilate because they are filled like skins But the contrary is obvious in arteriotomy and in wounds, for the blood spurting from the arteries escapes with force, now farther, now not so far, alternately, or in jets, and the jet always takes place with the diastole of the artery, never with the systole By which it clearly appears that the artery is dilated by the impulse of the blood, for of itself it would not throw the blood to such a distance, and whilst it was dilating, it ought rather to draw air into its cavity through the wound, were those things true that are commonly stated concerning the uses of the arteries Nor let the thickness of the arterial tunics impose upon us, and lead us to conclude that the pulsative property proceeds along them from the heart For in several animals the arteries do not apparently differ from the veins, and in extreme parts of the body, where the arteries are minutely subdivided, as in the brain, the hand, etc, no one could distinguish the arteries from the veins by the dissimilar characters of their coats, the tunics of both are identical And then, in an aneurism proceeding from a wounded or eroded artery, the pulsation is precisely the same as in the other arteries, and yet it has no proper arterial tunic This the learned Riolanus testifies to, along with me, in his Seventh Book

Nor let any one imagine that the uses of the pulse and the respiration are the same, because under the influence of the same causes, such as running, anger, the warm bath, or any other heating thing, as Galen says, they become more frequent and forcible together For, not only is experience in opposition to this idea, though Galen endeavors to explain it away, when we see that with excessive repletion the pulse beats more forcibly, whilst the respiration is diminished in amount, but in young persons the pulse is quick, while respiration is slow So also is it in alarm, and amidst care, and under anxiety of mind, sometimes, too, in fevers, the pulse is rapid, but the respiration is slower than usual

These and other objections of the same kind may be urged against the opinions mentioned Nor are the views that are entertained of the offices and pulse of the heart, perhaps, less bound up with great and most inextricable difficulties The heart, it is vulgarly said, is the fountain and workshop of the vital spirits, the centre from whence life is dispensed to the several parts of the body, and yet it is denied that the right ventricle makes spirits, it is rather held to supply nourishment to the lungs, whence it is maintained that fishes are without any right ventricle (and indeed every animal wants a right ventricle which is unfurnished with lungs), and that the right ventricle is present solely for the sake of the lungs

1 Why, I ask, when we see that the structure of both ventricles is almost identical, there being the same apparatus of fibres, and braces, and valves, and vessels, and auricles, and in both the same infarction of blood, in the subjects of our dissections, of the like black colour, and coagulated—why, I say, should their uses be imagined to be different, when the action, motion, and pulse of both are the same? If the three tricuspid valves placed at the entrance into the right ventricle prove obstacles to the reflux of blood into the vena cava, and if the three semilunar valves, which are situated at the commencement of the pulmonary artery be there, that they may prevent the return of blood into the ventricle, wherefore, when we find similar structures in connexion with the left ventricle, should we deny that they are there for the same end, of preventing here the egress, there the regurgitation of the blood?

2 And again, when we see that these structures, in point of size, form, and situation, are almost in every respect the same in the left as in the right ventricle, wherefore should it be maintained that things are here arranged in connexion with the egress and regress of spirits, there, i e, in the right, of blood The same arrangement cannot be held fitted to favor or impede the motion of blood and of spirits indifferently

3 And when we observe that the passages and vessels are severally in relation to one another in point of size, viz, the pulmonary artery to the pulmonary veins, wherefore should the one be imagined destined to a private or particular purpose, that to wit, of nourishing the lungs the other to a public and general function?

4 And, as Realdus Columbus says, how can it be conceived that such a quantity of blood should be required for the nutrition of the lungs, the vessel that leads to them, the vena arteriosa or pulmonary artery being of greater capacity than both the iliac veins?

5 And I ask further, as the lungs are so close at hand, and in continual motion, and the vessel that supplies them is of such dimensions, what is the use or meaning of the pulse of the right ventricle? And why was nature reduced to the necessity of adding another ventricle for the sole purpose of nourishing the lungs?

When it is said that the left ventricle obtains materials for the formation of spirits, air to wit, and blood, from the lungs and right sinuses of the heart, and in like manner sends spiritous blood into the aorta, drawing fuliginous vapours from thence, and sending them by the arteria venosa into the lungs, whence spirits are at the same time obtained for transmission into the aorta, I ask how, and by what means, is the separation effected? And how comes it that spirits and fuliginous vapours can pass hither and thither without admixture or confusion? If the mitral cuspidate valves do not prevent the egress of fuliginous vapours to the lungs, how should they oppose the escape of air? And how should the semilunars hinder the regress of spirits from the aorta upon each supervening diastole of the heart? And, above all, how can they say that the spiritous blood is sent from the arteria venalis (pulmonary veins) by the left ventricle into the lungs without any obstacle to its passage from the mitral valves, when they have previously asserted that the air entered by the same vessel from the lungs into the left ventricle, and have brought forward these same mitral valves as obstacles to its retrogression? Good God! how should the mitral valves prevent regurgitation of air and not of blood?

Further, when they dedicate the vena arteriosa (or pulmonary artery), a vessel of great size, and having the tunics of an artery, to none but a kind of private or single purpose, that, namely, of nourishing the lungs, why should the arteria venalis (or pulmonary veins), which is scarcely of similar size, which has the coats of a vein, and is soft and lax, be presumed to be made for many—three or four, different uses? For they will have it that air passes through this vessel from the lungs into the left ventricle, that fuliginous vapours escape by it from the heart into the lungs, and that a portion of the spiritous or spiritualized blood is distributed by it to the lungs for their refreshment.

If they will have it that fumes and air—fumes flowing from, air proceeding towards the heart—are transmitted by the same conduit, I reply, that nature is not wont to institute but one vessel, to contrive but one way for such contrary motions and purposes, nor is anything of the kind seen elsewhere.

If fumes or fuliginous vapours and air permeate this vessel, as they do the pulmonary bronchia, wherefore do we find neither air nor fuliginous

vapours when we divide the arteria venosa? Why do we always find this vessel full of sluggish blood, never of air? Whilst in the lungs we find abundance of air remaining.

If any one will perform Galen's experiment of dividing the trachea of a living dog, forcibly distending the lungs with a pair of bellows, and then tying the trachea securely, he will find, when he has laid open the thorax, abundance of air in the lungs, even to their extreme investing tunic, but none in either pulmonary veins, or left ventricle of the heart. But did the heart either attract air from the lungs, or did the lungs transmit any air to the heart, in the living dog, by so much the more ought this to be the case in the experiment just referred to. Who, indeed, doubts that, did he inflate the lungs of a subject in the dissecting-room, he would instantly see the air making its way by this route, were there actually any such passage for it? But this office of the pulmonary veins, namely, the transference of air from the lungs to the heart, is held of such importance, that Hieronymus Fabricius, of Aquapendente, maintains the lungs were made for the sake of this vessel, and that it constitutes the principal element in their structure.

But I should like to be informed wherefore, if the pulmonary vein were destined for the conveyance of air, it has the structure of a blood-vessel here. Nature had rather need of annular tubes, such as those of the bronchia, in order that they might always remain open, not have been liable to collapse, and that they might continue entirely free from blood, lest the liquid should interfere with the passage of the air, as it so obviously does when the lungs labour from being either greatly oppressed or loaded in a less degree with phlegm, as they are when the breathing is performed with a sibilous or rattling noise.

Still less is that opinion to be tolerated which (as a two-fold matter, one aereal, one sanguineous, is required for the composition of vital spirits) supposes the blood to ooze through the septum of the heart from the right to the left ventricle by certain secret pores, and the air to be attracted from the lungs through the great vessel, the pulmonary vein, and which will have it consequently, that there are numerous pores in the septum cordis adapted for the transmission of the blood. But, in faith, no such pores can be demonstrated, neither in fact do any such exist. For the septum of the heart is of a denser and more compact structure than any portion of the body, except the bones and sinews. But even supposing that there were foramina or pores in this situation, how could one of the ventricles extract anything from the other—the left, e.g., obtain blood from the right, when we see that both ventricles contract and dilate simultaneously? Wherefore should we not rather believe that the right took spirits from the left, than that the left obtained blood from the right ventricle, through these foramina? But it is certainly mysterious and incongruous that blood should be supposed to be most commodiously drawn through a set of obscure or invisible pores,

and air through perfectly open passages, at one and the same moment And why, I ask, is recourse had to secret and invisible porosities, to uncertain and obscure channels, to explain the passage of the blood into the left ventricle, when there is so open a way through the pulmonary veins? I own it has always appeared extraordinary to me that they should have chosen to make, or rather to imagine, a way through the thick, hard, and extremely compact substance of the septum cordis, rather than to take that by the open vas venosum or pulmonary vein, or even through the lax, soft and spongy substance of the lungs at large Besides, if the blood could permeate the substance of the septum, or could be imbibed from the ventricles, what use were there for the coronary artery and vein, branches of which proceed to the septum itself, to supply it with nourishment? And what is especially worthy of notice is this if in the foetus, where everything is more lax and soft, nature saw herself reduced to the necessity of bringing the blood from the right into the left side of the heart by the foramen ovale, from the vena cava through the arteria venosa, how should it be likely that in the adult she should pass it so commodiously, and without an effort, through the septum ventriculorum, which has now become denser by age?

Andreas Laurentius,[1] resting on the authority of Galen[2] and the experience of Hollerius, asserts and proves that the serum and pus in empyema, absorbed from the cavities of the chest into the pulmonary vein, may be expelled and got rid of with the urine and faeces through the left ventricle of the heart and arteries He quotes the case of a certain person affected with melancholia, and who suffered from repeated fainting fits who was relieved from the paroxysms on passing a quantity of turbid, fetid, and acrid urine, but he died at last, worn out by the disease, and when the body came to be opened after death, no fluid like that he had micturated was discovered either in the bladder or in the kidneys, but in the left ventricle of the heart and cavity of the thorax plenty of it was met with, and then Laurentius boasts that he had predicted the cause of the symptoms For my own part, however, I cannot but wonder, since he had divined and predicted that heterogenous matter could be discharged by the course he indicates, why he could not or would not perceive, and inform us that, in the natural state of things, the blood might be commodiously transferred from the lungs to the left ventricle of the heart by the very same route

Since, therefore, from the foregoing considerations and many others to the same effect, it is plain that what has heretofore been said concerning the motion and function of the heart and arteries must appear obscure, or inconsistent or even impossible to him who carefully considers the entire subject, it will be proper to look more narrowly into the matter, to contemplate the motion of the heart and arteries, not only in man, but

[1] Lib ix, cap xi quest 12

[2] De Locis Affectis lib vi cap 7

in all animals that have hearts, and further, by frequent appeals to vivisection, and constant ocular inspection, to investigate and endeavor to find the truth.

CHAPTER I

THE AUTHOR'S MOTIVES FOR WRITING

When I first gave my mind to vivisections, as a means of discovering the motions and uses of the heart, and sought to discover these from actual inspection, and not from the writings of others, I found the task so truly arduous, so full of difficulties, that I was almost tempted to think, with Fracastorius, that the motion of the heart was only to be comprehended by God. For I could neither rightly perceive at first when the systole and when the diastole took place, nor when and where dilatation and contraction occurred, by reason of the rapidity of the motion, which in many animals is accomplished in the twinkling of an eye, coming and going like a flash of lightning, so that the systole presented itself to me now from this point, now from that, the diastole the same, and then everything was reversed, the motions occurring, as it seemed, variously and confusedly together. My mind was therefore greatly unsettled, nor did I know what I should myself conclude, nor what believe from others, I was not surprised that Andreas Laurentius should have said that the motion of the heart was as perplexing as the flux and reflux of Euripus had appeared to Aristotle.

At length, and by using greater and daily diligence, having frequent recourse to vivisections, employing a variety of animals for the purpose, and collating numerous observations, I thought that I had attained to the truth, that I should extricate myself and escape from this labyrinth, and that I had discovered what I so much desired, both the motion and the use of the heart and arteries, since which time I have not hesitated to expose my views upon these subjects, not only in private to my friends, but also in public, in my anatomical lectures, after the manner of the Academy of old.

These views, as usual, pleased some more, others less, some chid and calumniated me, and laid it to me as a crime that I had dared to depart from the precepts and opinion of all anatomists, others desired further explanations of the novelties, which they said were both worthy of consideration, and might perchance be found of signal use. At length, yielding to the requests of my friends, that all might be made participators in my labours, and partly moved by the envy of others, who, receiving my views with uncandid minds and understanding them indifferently, have essayed to traduce me publicly, I have been moved to commit these things to the press, in order that all may be enabled to form an opinion both of me and my labours. This step I take all the more willingly, seeing that Hieronymus Fabricius of Aquapendente, although he has accurately and learnedly

delineated almost every one of the several parts of animals in a special work, has left the heart alone untouched Finally, if any use or benefit to this department of the republic of letters should accrue from my labours, it will, perhaps, be allowed that I have not lived idly, and, as the old man in the comedy says

> For never yet hath any one attained
> To such perfection, but that time, and place,
> And use, have brought addition to his knowledge,
> Or made correction, or admonished him,
> That he was ignorant of much which he
> Had thought he knew, or led him to reject
> What he had once esteemed of highest price

So will it, perchance, be found with reference to the heart at this time, or others, at least, starting from hence, the way pointed out to them, advancing under the guidance of a happier genius, may make occasion to proceed more fortunately, and to inquire more accurately

CHAPTER II

OF THE MOTIONS OF THE HEART, AS SEEN IN THE DISSECTION OF LIVING ANIMALS

In the first place, then, when the chest of a living animal is laid open and the capsule that immediately surrounds the heart is slit up or removed, the organ is seen now to move, now to be at rest,—there is a time when it moves, and a time when it is motionless

These things are more obvious in the colder animals, such as toads, frogs, serpents, small fishes, crabs, shrimps, snails and shell-fish They also become more distinct in warm-blooded animals, such as the dog and the hog, if they be attentively noted when the heart begins to flag, to move more slowly, and, as it were, to die the movements then become slower and rarer, the pauses longer, by which it is made much more easy to perceive and unravel what the motions really are, and how they are performed In the pause, as in death, the heart is soft, flaccid, exhausted, lying, as it were, at rest

In the motion, and interval in which this is accomplished, three principal circumstances are to be noted

1 That the heart is erected, and rises upward to a point, so that at this time it strikes against the breast and the pulse is felt externally

2 That it is everywhere contracted, but more especially towards the sides, so that it looks narrower, relatively longer, more drawn together The heart of an eel taken out of the body of the animal and placed upon the table or the hand, shows these particulars, but the same things are manifest in the heart of small fishes and of those colder animals where the organ is more conical or elongated

3 The heart being grasped in the hand, is felt to become harder during its action Now this hardness proceeds from tension, precisely as when the forearm is grasped, its tendons are perceived to become tense and resilient when the fingers are moved

4 It may further be observed in fishes, and the colder blooded animals, such as frogs, serpents, etc , that the heart, when it moves, becomes of a paler colour, when quiescent of a deeper blood-red colour

From these particulars it appeared evident to me that the motion of the heart consists in a certain universal tension—both contraction in the line of its fibres, and constriction in every sense It becomes erect, hard, and of diminished size during its action, the motion is plainly of the same nature as that of the muscles when they contract in the line of their sinews and fibres, for the muscles, when in action, acquire vigour and tenseness, and from soft become hard, prominent and thickened in the same manner the heart

We are therefore authorized to conclude that the heart, at the moment of its action, is at once constricted on all sides, rendered thicker in its parietes and smaller in its ventricles, and so made apt to project or expel its charge of blood This, indeed, is made sufficiently manifest by the fourth observation preceding in which we have seen that the heart, by squeezing out the blood it contains becomes paler, and then when it sinks into repose and the ventricle is filled anew with blood, that the deeper crimson colour returns But no one need rémain in doubt of the fact, for if the ventricle be pierced the blood will be seen to be forcibly projected outwards upon each motion or pulsation when the heart is tense

These things, therefore, happen together or at the same instant the tension of the heart, the pulse of its apex, which is felt externally by its striking against the chest, the thickening of its parietes, and the forcible expulsion of the blood it contains by the constriction of its ventricles

Hence the very opposite of the opinions commonly received, appears to be true, inasmuch as it is generally believed that when the heart strikes the breast and the pulse is felt without, the heart is dilated in its ventricles and is filled with blood, but the contrary of this is the fact, and the heart, when it contracts [and the shock is given], is emptied Whence the motion which is generally regarded as the diastole of the heart, is in truth its systole And in like manner the intrinsic motion of the heart is not the diastole but the systole, neither is it in the diastole that the heart grows firm and tense, but in the systole, for then only, when tense, is it moved and made vigorous

Neither is it by any means to be allowed that the heart only moves in the line of its straight fibres, although the great Vesalius, giving this notion countenance, quotes a bundle of osiers bound into a pyramidal heap in illustration, meaning, that as the apex is approached to the base, so are the sides made to bulge out in the fashion of arches, the cavities to

dilate, the ventricles to acquire the form of a cupping-glass and so to suck in the blood But the true effect of every one of its fibres is to constringe the heart at the same time that they render it tense, and this rather with the effect of thickening and amplifying the walls and substance of the organ than enlarging its ventricles And, again, as the fibres run from the apex to the base, and draw the apex towards the base, they do not tend to make the walls of the heart bulge out in circles, but rather the contrary, inasmuch as every fibre that is circularly disposed tends to become straight when it contracts, and is distended laterally and thickened, as in the case of muscular fibres in general, when they contract, that is when they are shortened longitudinally, as we see them in the bellies of the muscles of the body at large To all this let it be added that not only are the ventricles contracted in virtue of the direction and condensation of their walls, but farther, that those fibres, or bands, styled nerves by Aristotle, which are so conspicuous in the ventricles of the larger animals, and contain all the straight fibers (the parietes of the heart containing only circular ones) when they contract simultaneously, by an admirable adjustment all the internal surfaces are drawn together, as if with cords, and so is the charge of blood expelled with force

Neither is it true, as vulgarly believed, that the heart by any dilatation or motion of its own, has the power of drawing the blood into the ventricles, for when it acts and becomes tense, the blood is expelled, when it relaxes and sinks together it receives the blood in the manner and wise which will by and by be explained

CHAPTER III

OF THE MOTIONS OF ARTERIES, AS SEEN IN THE DISSECTION OF LIVING ANIMALS

In connexion with the motions of the heart these things are further to be observed having reference to the motions and pulses of the arteries

1 At the moment the heart contracts, and when the breast is struck, when in short the organ is in its state of systole, the arteries are dilated, yield a pulse, and are in the state of diastole In like manner, when the right ventricle contracts and propels its charge of blood, the arterial vein [the pulmonary artery] is distended at the same time with the other arteries of the body

2 When the left ventricle ceases to act, to contract, to pulsate, the pulse in the arteries also ceases, further, when this ventricle contracts languidly, the pulse in the arteries is scarcely perceptible In like manner, the pulse in the right ventricle failing, the pulse in the vena arteriosa [pulmonary artery] ceases also

3 Further, when an artery is divided or punctured, the blood is seen to be forcibly propelled from the wound at the moment the left ventricle contracts, and, again, when the pulmonary artery is wounded, the blood will be seen spouting forth with violence at the instant when the right ventricle contracts

So also in fishes, if the vessel which leads from the heart to the gills be divided, at the moment when the heart becomes tense and contracted, at the same moment does the blood flow with force from the divided vessel

In the same way, finally, when we see the blood in arteriotomy projected now to a greater, now to a less distance, and that the greater jet corresponds to the diastole of the artery and to the time when the heart contracts and strikes the ribs, and is in its state of systole, we understand that the blood is expelled by the same movement

From these facts, it is manifest, in opposition to commonly received opinions, that the diastole of the arteries corresponds with the time of the heart's systole, and that the arteries are filled and distended by the blood forced into them by the contraction of the ventricles, the arteries, therefore, are distended, because they are filled like sacs or bladders, and are not filled because they expand like bellows It is in virtue of one and the same cause, therefore, that all the arteries of the body pulsate, viz, the contraction of the left ventricle, in the same way as the pulmonary artery pulsates by the contraction of the right ventricle

Finally, that the pulses of the arteries are due to the impulses of the blood from the left ventricle, may be illustrated by blowing into a glove, when the whole of the fingers will be found to become distended at one and the same time, and in their tension to bear some resemblance to the pulse For in the ratio of the tension is the pulse of the heart, fuller, stronger, more frequent as that acts more vigorously, still preserving the rhythm and volume, and order of the heart's contractions Nor is it to be expected that because of the motion of the blood, the time at which the contraction of the heart takes place, and that at which the pulse in an artery (especially a distant one) is felt, shall be otherwise than simultaneous it is here the same as in blowing up a glove or bladder, for in a plenum (as in a drum, a long piece of timber, etc) the stroke and the motion occur at both extremities at the same time Aristotle,[1] too, has said, "the blood of all animals palpitates within their veins (meaning the arteries), and by the pulse is sent everywhere simultaneously " And further,[2] "thus do all the veins pulsate together and by successive strokes, because they all depend upon the heart, and, as it is always in motion, so are they likewise always moving together, but by successive movements " It is well to observe with Galen, in this place, that the old philosophers called the arteries veins

[1]De Animal iii cap 9
[2]De Respirat cap 20

I happened upon one occasion to have a particular case under my care, which plainly satisfied me of this truth A certain person was affected with a large pulsating tumour on the right side of the neck, called an aneurism, just at that part where the artery descends into the axilla, produced by an erosion of the artery itself, and daily increasing in size this tumour was visibly distended as it received the charge of blood brought to it by the artery, with each stroke of the heart the connexion of parts was obvious when the body of the patient came to be opened after his death The pulse in the corresponding arm was small, in consequence of the greater portion of the blood being diverted into the tumour and so intercepted

Whence it appears that wherever the motion of the blood through the arteries is impeded, whether it be by compression or infarction, or interception, there do the remote divisions of the arteries beat less forcibly, seeing that the pulse of the arteries is nothing more than the impulse or shock of the blood in these vessels

CHAPTER IV

OF THE MOTION OF THE HEART AND ITS AURICLES, AS SEEN IN THE BODIES OF LIVING ANIMALS

Besides the motions already spoken of, we have still to consider those that appertain to the auricles

Caspar Bauhin and John Riolan,[1] most learned men and skilful anatomists, inform us from their observations, that if we carefully watch the movements of the heart in the vivisection of an animal, we shall perceive four motions distinct in time and place, two of which are proper to the auricles, two to the ventricles With all deference to such authority I say, that there are four motions distinct in point of place, but not of time, for the two auricles move together, and so also do the two ventricles, in such wise that though the places be four, the times are only two And this occurs in the following manner

There are, as it were, two motions going on together, one of the auricles, another of the ventricles, these by no means taking place simultaneously, but the motion of the auricles preceding, that of the heart itself following, the motion appearing to begin from the auricles and to extend to the ventricles When all things are becoming languid, and the heart is dying, as also in fishes and the colder blooded animals, there is a short pause between these two motions, so that the heart aroused, as it were, appears to respond to the motion, now more quickly, now more tardily, and at length, and when near to death, it ceases to respond by its proper motion, but seems, as it were, to nod the head, and is so obscurely moved that it appears to give signs of motion to the pulsating

[1]Bauhin, lib ii, cap 21 Riolan lib viii, cap 1

auricle, rather than actually to move The heart, therefore, ceases to pulsate sooner than the auricles, so that the auricles have been said to outlive it, the left ventricle ceasing to pulsate first of all then its auricle, next the right ventricle, and, finally, all the other parts being at rest and dead, as Galen long since observed, the right auricle still continues to beat, life, therefore, appears to linger longest in the right auricle Whilst the heart is gradually dying, it is sometimes seen to reply, after two or three contractions of the auricles, roused as it were to action, and making a single pulsation, slowly, unwillingly, and with an effort

But this especially is to be noted, that after the heart has ceased to beat, the auricles however still contracting, a finger placed upon the ventricles perceives the several pulsations of the auricles, precisely in the same way and for the same reason, as we have said, that the pulses of the ventricles are felt in the arteries, to wit, the distension produced by the jet of blood And if at this time, the auricles alone pulsating, the point of the heart be cut off with a pair of scissors, you will perceive the blood flowing out upon each contraction of the auricles Whence it is manifest how the blood enters the ventricles, not by any attraction or dilatation of the heart, but thrown into them by the pulses of the auricles

And here I would observe, that whenever I speak of pulsations as occurring in the auricles or ventricles, I mean contractions first the auricles *contract,* and then and subsequently the heart itself *contracts* When the auricles contract they are seen to become whiter, especially where they contain but little blood, but they are filled as magazines or reservoirs of the blood, which is tending spontaneously and, by the motion of the veins, under pressure towards the centre, the whiteness indicated is most conspicuous towards the extremities or edges of the auricles at the time of their contractions

In fishes and frogs, and other animals which have hearts with but a single ventricle, and for an auricle have a kind of bladder much distended with blood, at the base of the organ, you may very plainly perceive this bladder contracting first, and the contraction of the heart or ventricle following afterwards

But I think it right to describe what I have observed of an opposite character the heart of an eel, of several fishes, and even of some [of the higher] animals taken out of the body, beats without auricles, nay, if it be cut in pieces the several parts may still be seen contracting and relaxing, so that in these creatures the body of the heart may be seen pulsating, palpitating, after the cessation of all motion in the auricle But is not this perchance peculiar to animals more tenacious of life, whose radical moisture is more glutinous, or fat and sluggish, and less readily soluble? The same faculty indeed appears in the flesh of eels, generally, which even when skinned and embowelled, and cut into pieces, are still seen to move

Experimenting with a pigeon upon one occasion, after the heart had wholly ceased to pulsate, and the auricles too had become motionless, I kept my finger wetted with saliva and warm for a short time upon the heart, and observed, that under the influence of this fomentation it recovered new strength and life, so that both ventricles and auricles pulsated, contracting and relaxing alternately, recalled as it were from death to life

Besides this, however I have occasionally observed, after the heart and even its right auricle had ceased pulsating,—when it was in articulo mortis in short, that an obscure motion, an undulation or palpitation, remained in the blood itself, which was contained in the right auricle, this being apparent so long as it was imbued with heat and spirit And indeed a circumstance of the same kind is extremely manifest in the course of the generation of animals as may be seen in the course of the first seven days of the incubation of the chick A drop of blood makes its appearance which palpitates as Aristotle had already observed, from this, when the growth is further advanced and the chick is fashioned, the auricles of the heart are formed, which pulsating henceforth give constant signs of life When at length, and after the lapse of a few days, the outline of the body begins to be distinguished, then is the ventricular part of the heart also produced, but it continues for a time white and apparently bloodless, like the rest of the animal, neither does it pulsate or give signs of motion I have seen a similar condition of the heart in the human foetus about the beginning of the third month, the heart being then whitish and bloodless, although its auricles contained a considerable quantity of purple blood In the same way in the egg, when the chick was formed and had increased in size, the heart too increased and acquired ventricles, which then began to receive and to transmit blood

And this leads me to remark, that he who inquires very particularly into this matter will not conclude that the heart, as a whole, is the primum vivens, ultimum moriens—the first part to live, the last to die, but rather its auricles, or the part which corresponds to the auricles in serpents, fishes, etc, which both lives before the heart[1] and dies after it

Nay, has not the blood itself or spirit an obscure palpitation inherent in it, which it has even appeared to me to retain after death? And it seems very questionable whether or not we are to say that life begins with the palpitation or beating of the heart The seminal fluid of all animals—the prolific spirit, as Aristotle observed, leaves their body with a bound and like a living thing, and nature in death, as Aristotle[2] further remarks, retracing her steps, reverts to whence she had set out, returns at the end of her course to the goal whence she had started, and as animal

[1]The reader will observe that Harvey when he speaks of the *heart*, always means the ventricles or ventricular portion of the organ [Willis 1847]

[2]De Motu Animal cap 8

generation proceeds from that which is not animal, entity from non-entity, so, by a retrograde course, entity, by corruption, is resolved into non-entity, whence that in animals, which was last created, fails first, and that which was first, fails last

I have also observed, that almost all animals have truly a heart, not the larger creatures only and those that have red blood, but the smaller, and [seemingly] bloodless ones also, such as slugs, snails, scallops, shrimps, crabs, crayfish, and many others, nay, even in wasps, hornets and flies, I have, with the aid of a magnifying glass, and at the upper part of what is called the tail, both seen the heart pulsating myself, and shown it to many others

But in the exsanguine tribes the heart pulsates sluggishly and deliberately, contracting slowly as in animals that are moribund, a fact that may readily be seen in the snail, whose heart will be found at the bottom of that orifice in the right side of the body which is seen to be opened and shut in the course of respiration, and whence saliva is discharged, the incision being made in the upper aspect of the body, near the part which corresponds to the liver

This, however, is to be observed that in winter and the colder season, exsanguine animals, such as the snail, show no pulsations, they seem rather to live after the manner of vegetables, or of those other productions which are therefore designated plant-animals

It is also to be noted that all animals which have a heart, have also auricles, or something analogous to auricles, and further, that wherever the heart has a double ventricle there are always two auricles present, but not otherwise If you turn to the production of the chick in ovo, however, you will find at first no more than a vesicle or auricle, or pulsating drop of blood, it is only by and by, when the development has made some progress, that the heart is fashioned even so in certain animals not destined to attain to the highest perfection in their organization, such as bees, wasps, snails, shrimps, crayfish, etc, we only find a certain pulsating vesicle, like a sort of red or white palpitating point, as the beginning or principle of their life[1]

We have a small shrimp in these countries, which is taken in the Thames and in the sea, the whole of whose body is transparent, this creature, placed in a little water, has frequently afforded myself and particular friends an opportunity of observing the motions of the heart with the greatest distinctness, the external parts of the body presenting no obstacle to our view, but the heart being perceived as though it had been seen through a window

I have also observed the first rudiments of the chick in the course of the fourth or fifth day of the incubation in the guise of a little cloud, the

[1]The Editor begs here to be allowed to remark on Harvey's obvious perception of the correspondence between that permanent condition of an organ in the lower and its transitory condition in the higher animals [Willis 1847]

shell having been removed and the egg immersed in clear tepid water In the midst of the cloudlet in question there was a bloody point so small that it disappeared during the contraction and escaped the sight, but in the relaxation it reappeared again, red and like the point of a pin, so that betwixt the visible and invisible, betwixt being and not being, as it were, it gave by its pulses a kind of representation of the commencement of life[1]

CHAPTER V

OF THE MOTION, ACTION, AND OFFICE OF THE HEART

From these and other observations of the like kind, I am persuaded it will be found that the motion of the heart is as follows

First of all, the auricle contracts, and in the course of its contraction throws the blood (which it contains in ample quantity as the head of the veins, the store-house and cistern of the blood) into the ventricle, which being filled, the heart raises itself straightway, makes all its fibres tense, contracts the ventricles, and performs a beat, by which beat it immediately sends the blood supplied to it by the auricle into the arteries, the right ventricle sending its charge into the lungs by the vessel which is called vena arteriosa, but which, in structure and function, and all things else, is an artery, the left ventricle sending its charge into the aorta, and through this by the arteries to the body at large

These two motions, one of the ventricles, another of the auricles, take place consecutively, but in such a manner that there is a kind of harmony or rhythm preserved between them, the two concurring in such wise that but one motion is apparent, especially in the warmer blooded animals, in which the movements in question are rapid Nor is this for any other reason than it is in a piece of machinery, in which, though one wheel gives motion to another, yet all the wheels seem to move simultaneously, or in that mechanical contrivance which is adapted to firearms, where the trigger being touched, down comes the flint, strikes against the steel, elicits a spark, which falling among the powder, is ignited, upon which the flame extends, enters the barrel, causes the explosion, propels the ball and the mark is attained—all of which incidents, by reason of the celerity with which they happen, seem to take place in the twinkling of an eye So also in deglutition by the elevation of the root of the tongue, and the compression of the mouth, the food or drink is pushed into the fauces, the larynx is closed by its own muscles, and the epiglottis, whilst the pharynx, raised and opened by its muscles no otherwise than is a sac that is to be filled, is lifted up, and its mouth dilated, upon which, the mouthful being received, it is forced downwards by the

[1]At the period Harvey indicates a rudimentary auricle and ventricle exist, but are so transparent that unless with certain precautions their parietes cannot be seen The filling and emptying of them, therefore give the appearance of a speck of blood alternately appearing and disappearing [Willis, 1847]

transverse muscles, and then carried farther by the longitudinal ones. Yet are all these motions, though executed by different and distinct organs, performed harmoniously, and in such order, that they seem to constitute but a single motion and act, which we call deglutition.

Even so does it come to pass with the motions and action of the heart, which constitute a kind of deglutition, a transfusion of the blood from the veins to the arteries. And if any one, bearing these things in mind, will carefully watch the motions of the heart in the body of a living animal, he will perceive not only all the particulars I have mentioned, viz., the heart becoming erect, and making one continuous motion with its auricles, but farther, a certain obscure undulation and lateral inclination in the direction of the axis of the right ventricle [the organ], twisting itself slightly in performing its work. And indeed every one may see, when a horse drinks, that the water is drawn in and transmitted to the stomach at each movement of the throat, the motion being accompanied with a sound, and yielding a pulse both to the ear and the touch, in the same way it is with each motion of the heart, when there is the delivery of a quantity of blood from the veins to the arteries, that a pulse takes place, and can be heard within the chest.

The motion of the heart, then, is entirely of this description, and the one action of the heart is the transmission of the blood and its distribution, by means of the arteries, to the very extremities of the body, so that the pulse which we feel in the arteries is nothing more than the impulse of the blood derived from the heart.

Whether or not the heart, besides propelling the blood, giving it motion locally, and distributing it to the body, adds anything else to it,—heat, spirit, perfection,—must be inquired into by and by, and decided upon other grounds. So much may suffice at this time, when it is shown that by the action of the heart the blood is transfused through the ventricles from the veins to the arteries, and distributed by them to all parts of the body.

So much, indeed, is admitted by all [physiologists], both from the structure of the heart and the arrangement and action of its valves. But still they are like persons purblind or groping about in the dark, and then they give utterance to diverse, contradictory, and incoherent sentiments, delivering many things upon conjecture, as we have already had occasion to remark.

The grand cause of hesitation and error in this subject appears to me to have been the intimate connexion between the heart and the lungs. When men saw both the vena arteriosa [or pulmonary artery] and the arteriae venosae [or pulmonary veins] losing themselves in the lungs, of course it became a puzzle to them to know how or by what means the right ventricle should distribute the blood to the body, or the left draw it from the venae cavae. This fact is borne witness to by Galen, whose words, when writing

against Erasistratus in regard to the origin and use of the veins and the coction of the blood, are the following:[1] "You will reply," he says, "that the effect is so, that the blood is prepared in the liver, and is thence transferred to the heart to receive its proper form and last perfection, a statement which does not appear devoid of reason, for no great and perfect work is ever accomplished at a single effort, or receives its final polish from one instrument. But if this be actually so, then show us another vessel which draws the absolutely perfect blood from the heart, and distributes it as the arteries do the spirits over the whole body." Here then is a reasonable opinion not allowed, because, forsooth, besides not seeing the true means of transit, he could not discover the vessel which should transmit the blood from the heart to the body at large!

But had any one been there in behalf of Erasistratus, and of that opinion which we now espouse, and which Galen himself acknowledges in other respects consonant with reason, to have pointed to the aorta as the vessel which distributes the blood from the heart to the rest of the body, I wonder what would have been the answer of that most ingenious and learned man? Had he said that the artery transmits spirits and not blood, he would indeed sufficiently have answered Erasistratus, who imagined that the arteries contained nothing but spirits, but then he would have contradicted himself, and given a foul denial to that for which he had keenly contended in his writings against this very Erasistratus, to wit, that blood in substance is contained in the arteries, and not spirits, a fact which he demonstrated not only by many powerful arguments, but by experiments.

But if the divine Galen will here allow, as in other places he does, "that all the arteries of the body arise from the great artery, and that this takes its origin from the heart, that all these vessels naturally contain and carry blood, that the three semilunar valves situated at the orifice of the aorta prevent the return of the blood into the heart, and that nature never connected them with this, the most noble viscus of the body, unless for some most important end", if, I say, this father of physic admits all these things,—and I quote his own words,—I do not see how he can deny that the great artery is the very vessel to carry the blood, when it has attained its highest term of perfection, from the heart for distribution to all parts of the body. Or would he perchance still hesitate, like all who have come after him, even to the present hour, because he did not perceive the route by which the blood was transferred from the veins to the arteries, in consequence, as I have already said, of the intimate connexion between the heart and the lungs? And that this difficulty puzzled anatomists not a little, when in their dissections they found the pulmonary artery and left ventricle full of thick, black, and clotted blood, plainly appears, when they felt themselves compelled to

[1] De Placitis Hippocratis et Platonis vi.

affirm that the blood made its way from the right to the left ventricle by sweating through the septum of the heart. But this fancy I have already refuted. A new pathway for the blood must therefore be prepared and thrown open, and being once exposed, no further difficulty will, I believe, be experienced by anyone in admitting what I have already proposed in regard to the pulse of the heart and arteries, viz. the passage of the blood from the veins to the arteries, and its distribution to the whole of the body by means of these vessels.

CHAPTER VI

OF THE COURSE BY WHICH THE BLOOD IS CARRIED FROM THE VENA CAVA INTO THE ARTERIES, OR FROM THE RIGHT INTO THE LEFT VENTRICLE OF THE HEART

Since the intimate connexion of the heart with the lungs, which is apparent in the human subject, has been the probable cause of the errors that have been committed on this point, they plainly do amiss who, pretending to speak of the parts of animals generally, as anatomists for the most part do, confine their researches to the human body alone, and that when it is dead. They obviously act no otherwise than he who, having studied the forms of a single commonwealth, should set about the composition of a general system of polity, or who, having taken cognizance of the nature of a single field, should imagine that he had mastered the science of agriculture, or who, upon the ground of one particular proposition, should proceed to draw general conclusions.

Had anatomists only been as conversant with the dissection of the lower animals as they are with that of the human body, the matters that have hitherto kept them in a perplexity of doubt would, in my opinion, have met them freed from every kind of difficulty.

And, first, in fishes, in which the heart consists of but a single ventricle, they having no lungs, the thing is sufficiently manifest. Here the sac, which is situated at the base of the heart, and is the part analogous to the auricle in man, plainly throws the blood into the heart, and the heart, in its turn, conspicuously transmits it by a pipe or artery, or vessel analogous to an artery; these are facts which are confirmed by simple ocular inspection, as well as by a division of the vessel, when the blood is seen to be projected by each pulsation of the heart.

The same thing is also not difficult of demonstration in those animals that have either no more, or, as it were, no more than a single ventricle to the heart, such as toads, frogs, serpents, and lizards, which, although they have lungs in a certain sense, as they have a voice (and I have many observations by me on the admirable structure of the lungs of these

animals, and matters appertaining which, however, I cannot introduce in this place), still their anatomy plainly shows that the blood is transferred in them from the veins to the arteries in the same manner as in higher animals, viz., by the action of the heart; the way, in fact, is patent, open, manifest; there is no difficulty, no room for hesitating about it; for in them the matter stands precisely as it would in man, were the septum of his heart perforated or removed, or one ventricle made out of two; and this being the case, I imagine that no one will doubt as to the way by which the blood may pass from the veins into the arteries.

But as there are actually more animals which have no lungs than there are which be furnished with them, and in like manner a greater number which have only one ventricle than there are which have two, it is open to us to conclude, judging from the mass or multitude of living creatures, that for the major part, and generally, there is an open way by which the blood is transmitted from the veins through the sinuses or cavities of the heart into the arteries.

I have, however, cogitating with myself, seen further, that the same thing obtained most obviously in the embryos of those animals that have lungs; for in the foetus the four vessels belonging to the heart, viz., the vena cava, the vena arteriosa or pulmonary artery, the arteria venalis or pulmonary vein, and the arteria magna or aorta, are all connected otherwise than in the adult, a fact sufficiently known to every anatomist. The first contact and union of the vena cava with the arteria venosa or pulmonary veins, which occurs before the cava opens properly into the right ventricle of the heart, or gives off the coronary vein, a little above its escape from the liver, is by a lateral anastomosis; this is an ample foramen, of an oval form, communicating between the cava and the arteria venosa, or pulmonary vein, so that the blood is free to flow in the greatest abundance by that foramen from the vena cava into the arteria venosa or pulmonary vein, and left auricle, and from thence into the left ventricle; and farther, in this foramen ovale, from that part which regards the arteria venosa, or pulmonary vein, there is a thin tough membrane, larger than the opening, extended like an operculum or cover; this membrane in the adult blocking up the foramen, and adhering on all sides, finally closes it up, and almost obliterates every trace of it. This membrane, however, is so contrived in the foetus, that falling loosely upon itself, it permits a ready access to the lungs and heart, yielding a passage to the blood which is streaming from the cava, and hindering the tide at the same time from flowing back into that vein. All things, in short, permit us to believe that in the embryo the blood must constantly pass by this foramen from the vena cava into the arteria venosa, or pulmonary vein, and from thence into the left auricle of the heart; and having once entered there, it can never regurgitate.

Another union is that by the vena arteriosa, or pulmonary artery, and is effected when that vessel divides into two branches after its escape

from the right ventricle of the heart. It is as if to the two trunks already mentioned a third were superadded, a kind of arterial canal, carried obliquely from the vena arteriosa, or pulmonary artery, to perforate and terminate in the arteria magna or aorta. In the embryo, consequently, there are, as it were, two aortas, or two roots of the arteria magna, springing from the heart. This canalis arteriosus shrinks gradually after birth, and is at length and finally almost entirely withered, and removed, like the Umbilical vessels.

The canalis arteriosus contains no membrane or valve to direct or impede the flow of the blood in this or in that direction: for at the root of the vena arteriosa, or pulmonary artery, of which the canalis arteriosus is the continuation in the foetus, there are three sigmoid or semilunar valves, which open from within outwards, and oppose no obstacle to the blood flowing in this direction or from the right ventricle into the pulmonary artery and aorta, but they prevent all regurgitation from the aorta or pulmonic vessels back upon the right ventricle, closing with perfect accuracy, they oppose an effectual obstacle to everything of the kind in the embryo. So that there is also reason to believe that when the heart contracts, the blood is regularly propelled by the canal or passage indicated from the right ventricle into the aorta.

What is commonly said in regard to these two great communications, to wit, that they exist for the nutrition of the lungs, is both improbable and inconsistent, seeing that in the adult they are closed up, abolished, and consolidated, although the lungs, by reason of their heat and motion, must then be presumed to require a larger supply of nourishment. The same may be said in regard to the assertion that the heart in the embryo does not pulsate, that it neither acts nor moves, so that nature was forced to make these communications for the nutrition of the lungs. This is plainly false, for simple inspection of the incubated egg, and of embryos just taken out of the uterus, shows that the heart moves precisely in them as in adults, and that nature feels no such necessity. I have myself repeatedly seen these motions, and Aristotle is likewise witness of their reality. "The pulse," he observes, "inheres in the very constitution of the heart, and appears from the beginning, as is learned both from the dissection of living animals, and the formation of the chick in the egg."[1] But we further observe, that the passages in question are not only pervious up to the period of birth in man, as well as in other animals, as anatomists in general have described them, but for several months subsequently, in some indeed for several years, not to say for the whole course of life, as, for example, in the goose, snipe, and various birds, and many of the smaller animals. And this circumstance it was, perhaps, that imposed upon Botallus, who thought he had discovered a new passage for the blood from the vena cava into the left ventricle of the heart, and I own that when I met with the same arrangement in one of the

[1]Lib. de Spiritu, cap. v.

larger members of the mouse family, in the adult state, I was myself at first led to something of a like conclusion.

From this it will be understood that in the human embryo, and in the embryos of animals in which the communications are not closed, the same thing happens, namely, that the heart by its motion propels the blood by obvious and open passages from the vena cava into the aorta through the cavities of both the ventricles, the right one receiving the blood from the auricle, and propelling it by the vena arteriosa, or pulmonary artery, and its continuation, named the ductus arteriosus, into the aorta, the left, in like manner, charged by the contraction of its auricle, which has received its supply through the foramen ovale from the vena cava, contracting, and projecting the blood through the root of the aorta into the trunk of that vessel.

In embryos, consequently, whilst the lungs are yet in a state of inaction, performing no function, subject to no motion any more than if they had not been present, nature uses the two ventricles of the heart as if they formed but one, for the transmission of the blood. The condition of the embryos of those animals which have lungs, whilst these organs are yet in abeyance and not employed, is the same as that of those animals which have no lungs.

So clearly, therefore, does it appear in the case of the foetus, viz., that the heart by its action transfers the blood from the vena cava into the aorta, and that by a route as obvious and open, as if in the adult the two ventricles were made to communicate by the removal of their septum. Since, then, we find that in the greater number of animals, in all, indeed, at a certain period of their existence, the channels for the transmission of the blood through the heart are so conspicuous, we have still to inquire wherefore in some creatures—those, namely, that have warm blood, and that have attained to the adult age, man among the number—we should not conclude that the same thing is accomplished through the substance of the lungs, which in the embryo, and at a time when the function of these organs is in abeyance, nature effects by the direct passages described, and which, indeed, she seems compelled to adopt through want of a passage by the lungs, or wherefore it should be better (for nature always does that which is best) that she should close up the various open routes which she had formerly made use of in the embryo and foetus, and still uses in all other animals, not only opening up no new apparent channels for the passage of the blood, therefore, but even entirely shutting up those which formerly existed.

And now the discussion is brought to this point, that they who inquire into the ways by which the blood reaches the left ventricle of the heart and pulmonary veins from the vena cava, will pursue the wisest course if they seek by dissection to discover the causes why in the larger and more perfect animals of mature age, nature has rather chosen to make the

blood percolate the parenchyma of the lungs than as in other instances chosen a direct and obvious course—for I assume that no other path or mode of transit can be entertained It must be either because the larger and more perfect animals are warmer, and when adult their heat greater—ignited, as I might say, and requiring to be damped or mitigated, therefore it may be that the blood is sent through the lungs, that it may be tempered by the air that is inspired, and prevented from boiling up, and so becoming extinguished, or something else of the sort But to determine these matters, and explain them satisfactorily, were to enter on a speculation in regard to the office of the lungs and the ends for which they exist, and upon such a subject as well as upon what pertains to eventilation, to the necessity and use of the air, etc, as also to the variety and diversity of organs that exist in the bodies of animals in connexion with these matters, although I have made a vast number of observations, still, lest I should be held as wandering too wide of my present purpose, which is the use and motion of the heart, and be charged with speaking of things beside the question, and rather complicating and quitting than illustrating it, I shall leave such topics till I can more conveniently set them forth in a treatise apart And now, returning to my immediate subject, I go on with what yet remains for demonstration, viz, that in the more perfect and warmer adult animals, and man, the blood passes from the right ventricle of the heart by the vena arteriosa, or pulmonary artery, into the lungs, and thence by the arteriae venosae, or pulmonary veins, into the left auricle, and thence into the left ventricle of the heart And, first, I shall show that this may be so, and then I shall prove that it is so in fact

CHAPTER VII

THE BLOOD PERCOLATES THE SUBSTANCE OF THE LUNGS FROM THE RIGHT VENTRICLE OF THE HEART INTO THE PULMONARY VEINS AND LEFT VENTRICLE

That this is possible, and that there is nothing to prevent it from being so, appears when we reflect on the way in which water percolating the earth produces springs and rivulets, or when we speculate on the means by which the sweat passes through the skin, or the urine through the parenchyma of the kidneys It is well known that persons who use the Spa waters, or those of La Madonna, in the territories of Padua, or others of an acidulous or vitriolated nature, or who simply swallow drinks by the gallon, pass all off again within an hour or two by urine Such a quantity of liquid must take some short time in the concoction it must pass through the liver (it is allowed by all that the juices of the food we consume pass

twice through this organ in the course of the day), it must flow through the veins, through the parenchyma of the kidneys, and through the ureters into the bladder

To those, therefore, whom I hear denying that the blood, aye the whole mass of the blood may pass through the substance of the lungs, even as the nutritive juices percolate the liver, asserting such a proposition to be impossible, and by no means to be entertained as credible, I reply, with the poet, that they are of that race of men who, when they will, assent full readily, and when they will not, by no manner of means, who, when their assent is wanted, fear, and when it is not fear not to give it

The parenchyma of the liver is extremely dense, so is that of the kidney, the lungs, again, are of a much looser texture, and if compared with the kidneys are absolutely spongy In the liver there is no forcing, no impelling power, in the lungs the blood is forced on by the pulse of the right ventricle, the necessary effect of whose impulse is the distension of the vessels and pores of the lungs And then the lungs, in respiration, are perpetually rising and falling, motions, the effect of which must needs be to open and shut the pores and vessels, precisely as in the case of a sponge, and of parts having a spongy structure, when they are alternately compressed and again are suffered to expand The liver, on the contrary, remains at rest, and is never seen to be dilated and constricted Lastly, if no one denies the possibility of the whole of the ingested juices passing through the liver, in man, oxen, and the larger animals, generally, in order to reach the vena cava, and for this reason, that if nourishment is to go on, these juices must needs get into the veins, and there is no other way but the one indicated, why should not the same arguments be held of avail for the passage of the blood in adults through the lungs? Why not, with Columbus, that skillful and learned anatomist, maintain and believe the like, from the capacity and structure of the pulmonary vessels, from the fact of the pulmonary veins and ventricle corresponding with them, being always found to contain blood, which must needs have come from the veins, and by no other passage save through the lungs? Columbus, and we also, from what precedes, from dissections, and other arguments, conceive the thing to be clear But as there are some who admit nothing unless upon authority, let them learn that the truth I am contending for can be confirmed from Galen's own words, namely, that not only may the blood be transmitted from the pulmonary artery into the pulmonary veins, then into the left ventricle of the heart, and from thence into the arteries of the body, but that this is effected by the ceaseless pulsation of the heart and the motion of the lungs in breathing

There are, as every one knows, three sigmoid or semilunar valves situated at the orifice of the pulmonary artery, which effectually prevent the blood sent into the vessel from returning into the cavity of the heart Now Galen, explaining the uses of these valves, and the necessity for them, em-

ploys the following language[1] "There is everywhere a mutual anastomosis and inosculation of the arteries with the veins, and they severally transmit both blood and spirit, by certain invisible and undoubtedly very narrow passages Now if the mouth of the vena arteriosa, or pulmonary artery, had stood in like manner continually open, and nature had found no contrivance for closing it when requisite, and opening it again, it would have been impossible that the blood could ever have passed by the invisible and delicate mouths, during the contractions of the thorax, into the arteries, for all things are not alike readily attracted or repelled but that which is light is more readily drawn in, the instrument being dilated, and forced out again when it is contracted, than that which is heavy, and in like manner is anything drawn more rapidly along an ample conduit, and again driven forth, than it is through a narrow tube But when the thorax is contracted, the pulmonary veins, which are in the lungs, being driven inwardly, and powerfully compressed on every side, immediately force out some of the spirit they contain, and at the same time assume a certain portion of blood by those subtile mouths, a thing that could never come to pass were the blood at liberty to flow back into the heart through the great orifice of the pulmonary artery But its return through this great opening being prevented, when it is compressed on every side, a certain portion of it distils into the pulmonary veins by the minute orifices mentioned" And shortly afterwards, in the very next chapter, he says "The more the thorax contracts, the more it strives to force out the blood, the more exactly do these membranes (viz, the sigmoid valves) close up the mouth of the vessel, and suffer nothing to regurgitate' The same fact he has also alluded to in a preceding part of the tenth chapter "Were there no valves, a three-fold inconvenience would result, so that the blood would then perform this lengthened course in vain, it would flow inwards during the diastoles of the lungs, and fill all their arteries, but in the systoles, in the manner of the tide, it would ever and anon, like the Euripus, flow backwards and forwards by the same way, with a reciprocating motion, which would nowise suit the blood This, however, may seem a matter of little moment, but if it meantime appear that the function of respiration suffer, then I think it would be looked upon as no trifle, etc" And again, and shortly afterwards "And then a third inconvenience, by no means to be thought lightly of would follow were the blood moved backwards during the expirations had not our Maker instituted those supplementary membranes [the sigmoid valves]" Whence, in the eleventh chapter, he concludes "That they have all a common use (to wit, the valves) and that it is to prevent regurgitation or backward motion, each however having a proper function the one set drawing matters from the heart, and preventing their return the other drawing

[1]De Usu Partium lib vi cap 10

matters into the heart, and preventing their escape from it For nature never intended to distress the heart with needless labour, neither to bring aught into the organ which it had been better to have kept away, nor to take from it again aught which it was requisite should be brought Since, then, there are four orifices in all, two in either ventricle, one of these induces, the other educes ' And again he says "Farther, since there is one vessel, consisting of a simple tunic implanted in the heart, and another, having a double tunic extending from it (Galen is here speaking of the right side of the heart but I extend his observations to the left side also), a kind of reservoir had to be provided, to which both belonging, the blood should be drawn in by the one and sent out by the other "

This argument Galen adduces for the transit of the blood by the right ventricle from the vena cava into the lungs, but we can use it with still greater propriety, merely changing the terms for the passage of the blood from the veins through the heart into the arteries From Galen, however, that great man that father of physicians it clearly appears that the blood passes through the lungs from the pulmonary artery into the minute branches of the pulmonary veins urged to this both by the pulses of the heart and by the motions of the lungs and thorax that the heart moreover is incessantly receiving and expelling the blood by and from its ventricles, as from a magazine or cistern, and for this end is furnished with four sets of valves, two serving for the induction and two for the eduction of the blood lest, like the Euripus, it should be incommodiously sent hither and thither or flow back into the cavity which it should have quitted, or quit the part where its presence was required, and so the heart be oppressed with labour in vain, and the office of the lungs be interfered with [1] Finally, our position that the blood is continually passing from the right to the left ventricle from the vena cava into the aorta, through the porous structure of the lungs, plainly appears from this that since the blood is incessantly sent from the right ventricle into the lungs by the pulmonary artery, and in like manner is incessantly drawn from the lungs into the left ventricle, as appears from what precedes and the position of the valves, it cannot do otherwise than pass through continuously And then, as the blood is incessantly flowing into the right ventricle of the heart, and is continually passed out from the left, as appears in like manner, and as is obvious both to sense and reason, it is impossible that the blood can do otherwise than pass continually from the vena cava into the aorta

Dissection consequently shows distinctly what takes place [in regard to the transit of the blood] in the greater number of animals, and indeed in all, up to the period of their [foetal] maturity, and that the same thing occurs in adults is equally certain, both from Galen's words, and what has already been said on the subject, only that in the former

[1]See the Commentary of the learned Hofmann upon the Sixth Book of Galen De Usu Partium a work which I first saw after I had written what precedes

the transit is effected by open and obvious passages, in the latter by the obscure porosities of the lungs and the minute inosculations of vessels. Whence it appears that, although one ventricle of the heart, the left to wit, would suffice for the distribution of the blood over the body, and its eduction from the vena cava, as indeed is done in those creatures that have no lungs, nature, nevertheless, when she ordained that the same blood should also percolate the lungs, saw herself obliged to add another ventricle, the right, the pulse of which should force the blood from the vena cava through the lungs into the cavity of the left ventricle. In this way, therefore, it may be said that the right ventricle is made for the sake of the lungs, and for the transmission of the blood through them, not for their nutrition, seeing it were unreasonable to suppose that the lungs required any so much more copious a supply of nutriment, and that of so much purer and more spiritous a kind, as coming immediately from the ventricle of the heart, than either the brain with its peculiarly pure substance, or the eyes with their lustrous and truly admirable structure, or the flesh of the heart itself, which is more commodiously nourished by the coronary artery.

CHAPTER VIII

OF THE QUANTITY OF BLOOD PASSING THROUGH THE HEART FROM THE VEINS TO THE ARTERIES, AND OF THE CIRCULAR MOTION OF THE BLOOD

Thus far I have spoken of the passage of the blood from the veins into the arteries, and of the manner in which it is transmitted and distributed by the action of the heart, points to which some, moved either by the authority of Galen or Columbus, or the reasonings of others, will give in their adhesion. But what remains to be said upon the quantity and source of the blood which thus passes, is of so novel and unheard of character, that I not only fear injury to myself from the envy of a few, but I tremble lest I have mankind at large for my enemies, so much doth wont and custom, that become as another nature, and doctrine once sown and that hath struck deep root, and respect for antiquity influence all men. Still the die is cast, and my trust is in my love of truth, and the candour that inheres in cultivated minds. And sooth to say, when I surveyed my mass of evidence, whether derived from vivisections, and my various reflections on them, or from the ventricles of the heart, and the vessels that enter into and issue from them, the symmetry and size of these conduits—for nature doing nothing in vain, would never have given them so large a relative size without a purpose—or from the arrangement and intimate structure of the valves in particular, and of the other parts of the heart in general, with many things besides, I frequently and seriously bethought me, and long revolved in my mind, what might be the quantity of blood which was transmitted, in how short a time its

passage might be effected and the like, and not finding it possible that this could be supplied by the juices of the ingested aliment without the veins on the one hand becoming drained, and the arteries on the other getting ruptured through the excessive charge of blood, unless the blood should somehow find its way from the arteries into the veins, and so return to the right side of the heart, I began to think whether there might not be A MOTION, AS IT WERE, IN A CIRCLE Now this I afterwards found to be true, and I finally saw that the blood, forced by the action of the left ventricle into the arteries, was distributed to the body at large, and its several parts, in the same manner as it is sent through the lungs, impelled by the right ventricle into the pulmonary artery, and that it then passed through the veins and along the vena cava and so round to the left ventricle in the manner already indicated Which motion we may be allowed to call circular, in the same way as Aristotle says that the air and the rain emulate the circular motion of the superior bodies, for the moist earth, warmed by the sun evaporates the vapours drawn upwards are condensed and descending in the form of rain, moisten the earth again and by this arrangement are generations of living things produced, and in like manner too are tempests and meteors engendered by the circular motion, and by the approach and recession of the sun

And so, in all likelihood, does it come to pass in the body through the motion of the blood, the various parts are nourished, cherished quickened by the warmer, more perfect, vaporous spirituous and, as I may say, alimentive blood, which on the contrary, in contact with these parts becomes cooled, coagulated, and, so to speak, effete, whence it returns to its sovereign the heart, as if to its source, or to the inmost home of the body, there to recover its state of excellence or perfection Here it resumes its due fluidity and receives an infusion of natural heat —powerful, fervid, a kind of treasury of life, and is impregnated with spirits, and it might be said with balsam, and thence it is again dispersed, and all this depends on the motion and action of the heart

The heart, consequently, is the beginning of life the sun of the microcosm, even as the sun in his turn might well be designated the heart of the world, for it is the heart by whose virtue and pulse the blood is moved, perfected, made apt to nourish, and is preserved from corruption and coagulation, it is the household divinity which, discharging its function, nourishes, cherishes, quickens the whole body, and is indeed the foundation of life, the source of all action But of these things we shall speak more opportunely when we come to speculate upon the final cause of this motion of the heart

Hence, since the veins are the conduits and vessels that transport the blood, they are of two kinds, the cava and the aorta, and this not by reason of there being two sides of the body, as Aristotle has it, but be-

cause of the difference of office, nor yet, as is commonly said, in consequence of any diversity of structure, for in many animals, as I have said, the vein does not differ from the artery in the thickness of its tunics, but solely in virtue of their several destinies and uses A vein and an artery, both styled vein by the ancients, and that not undeservedly, as Galen has remarked, because the one, the artery to wit, is the vessel which carries the blood from the heart to the body at large, the other or vein of the present day bringing it back from the general system to the heart, the former is the conduit from, the latter the channel to, the heart, the latter contains the cruder, effete blood, rendered unfit for nutrition, the former transmits the digested, perfect, peculiarly nutritive fluid

CHAPTER IX

THAT THERE IS A CIRCULATION OF THE BLOOD IS CONFIRMED FROM THE FIRST PROPOSITION

But lest anyone should say that we give them words only, and make mere specious assertions without any foundation, and desire to innovate without sufficient cause, three points present themselves for confirmation, which being stated, I conceive that the truth I contend for will follow necessarily, and appear as a thing obvious to all First,—the blood is incessantly transmitted by the action of the heart from the vena cava to the arteries in such quantity, that it cannot be supplied from the ingesta, and in such wise that the whole mass must very quickly pass through the organ, second,—the blood under the influence of the arterial pulse enters and is impelled in a continuous, equable, and incessant stream through every part and member of the body, in much larger quantity than were sufficient for nutrition, or than the whole mass of fluids could supply, third, —the veins in like manner return this blood incessantly to the heart from all parts and members of the body These points proved, I conceive it will be manifest that the blood circulates, revolves, propelled and then returning, from the heart to the extremities, from the extremities to the heart, and thus that it performs a kind of circular motion

Let us assume either arbitrarily or from experiment, the quantity of blood which the left ventricle of the heart will contain when distended to be, say two ounces, three ounces, one ounce and a half—in the dead body I have found it to hold upwards of two ounces Let us assume further, how much less the heart will hold in the contracted than in the dilated state, and how much blood it will project into the aorta upon each contraction,—and all the world allows that with the systole something is always projected, a necessary consequence demonstrated in the third chapter, and obvious from the structure of the valves, and let us suppose as approaching the truth that the fourth, or fifth, or sixth, or even but the eighth part of its charge is thrown into the artery at each

contraction, this would give either half an ounce or three drachms, or one drachm of blood as propelled by the heart at each pulse into the aorta, which quantity, by reason of the valves at the root of the vessel, can by no means return into the ventricle Now in the course of half an hour, the heart will have made more than one thousand beats, in some as many as two, three, and even four thousand Multiplying the number of drachms propelled by the number of pulses, we shall have either one thousand half ounces, or one thousand times three drachms, or a like proportional quantity of blood according to the amount which we assume as propelled with each stroke of the heart sent from this organ into the artery, a larger quantity in every case than is contained in the whole body! In the same way in the sheep or dog, say that but a single scruple of blood passes with each stroke of the heart, in one-half hour we should have one thousand scruples, or about three pounds and a half of blood injected into the aorta, but the body of neither animal contains above four pounds of blood, a fact which I have myself ascertained in the case of the sheep

Upon this supposition, therefore, assumed merely as a ground for reasoning, we see the whole mass of blood passing through the heart, from the veins to the arteries, and in like manner through the lungs

But let it be said that this does not take place in half an hour, but in an hour, or even in a day, any way it is still manifest that more blood passes through the heart in consequence of its action, than can either be supplied by the whole of the ingesta, or than can be contained in the veins at the *same moment*

Nor can it be allowed that the heart in contracting sometimes propels and sometimes does not propel, or at most propels but very little, a mere nothing, or an imaginary something all this, indeed, has already been refuted, and is, besides, contrary both to sense and reason For if it be a necessary effect of the dilatation of the heart that its ventricles become filled with blood, it is equally so that, contracting, these cavities should expel their contents, and this is not in any trifling measure, seeing that neither are the conduits small, nor the contractions few in number, but frequent, and always in some certain proportion, whether it be a third or sixth, or an eighth, to the total capacity of the ventricles, so that a like proportion of blood must be expelled, and a like proportion received with each stroke of the heart, the capacity of the ventricle contracted always bearing a certain relation to the capacity of the ventricle when dilated And since in dilating, the ventricles cannot be supposed to get filled with nothing, or with an imaginary something, so in contracting they never expel nothing or aught imaginary, but always a certain something, viz, blood, in proportion to the amount of the contraction Whence it is to be inferred that if at one stroke the heart in man, the ox or the sheep, ejects but a single drachm of blood, and there are one thousand strokes in half an

hour, in this interval there will have been ten pounds five ounces expelled were there with each stroke two drachms expelled, the quantity would of course amount to twenty pounds and ten ounces, were there half an ounce, the quantity would come to forty-one pounds and eight ounces, and were there one ounce it would be as much as eighty-three pounds and four ounces, the whole of which, in the course of one half-hour, would have been transfused from the veins to the arteries. The actual quantity of blood expelled at each stroke of the heart, and the circumstances under which it is either greater or less than ordinary, I leave for particular determination afterwards, from numerous observations which I have made on the subject.

Meantime this much I know, and would here proclaim to all, that the blood is transfused at one time in larger, at another in smaller quantity, and that the circuit of the blood is accomplished now more rapidly, now more slowly, according to the temperament, age, etc., of the individual, to external and internal circumstances, to naturals and non-naturals,—sleep, rest, food, exercise, affections of the mind, and the like. But indeed, supposing even the smallest quantity of blood to be passed through the heart and lungs with each pulsation, a vastly greater amount would still be thrown into the arteries and whole body, than could by any possibility be supplied by the food consumed, in short it could be furnished in no other way than by making a circuit and returning.

This truth, indeed, presents itself obviously before us when we consider what happens in the dissection of living animals, the great artery need not divide, but a very small branch only (as Galen even proves in regard to man), to have the whole of the blood in the body, as well that of the veins as of the arteries, drained away in the course of no long time—some half-hour or less. Butchers are well aware of the fact and can bear witness to it, for, cutting the throat of an ox and so dividing the vessels of the neck, in less than a quarter of an hour they have all the vessels bloodless—the whole mass of blood has escaped. The same thing also occasionally occurs with great rapidity in performing amputations and removing tumours in the human subject.

Nor would this argument lose any of its force, did any one say that in killing animals in the shambles, and performing amputations, the blood escaped in equal, if not perchance in larger quantity by the veins than by the arteries. The contrary of this statement, indeed, is certainly the truth, the veins, in fact, collapsing, and being without any propelling power, and further, because of the impediment of the valves, as I shall show immediately, pour out but very little blood, whilst the arteries spout it forth with force abundantly, impetuously, and as if it were propelled by a syringe. And then the experiment is easily tried of leaving the vein untouched, and only dividing the artery in the neck of a sheep or dog, when it will be seen with what force, in what abundance,

and how quickly, the whole blood in the body, of the veins as well as of the arteries, is emptied But the arteries receive blood from the veins in no other way than by transmission through the heart, as we have already seen, so that if the aorta be tied at the base of the heart, and the carotid or any other artery be opened, no one will now be surprised to find it empty, and the veins only replete with blood

And now the cause is manifest, wherefore in our dissections we usually find so large a quantity of blood in the veins, so little in the arteries, wherefore there is much in the right ventricle, little in the left, circumstances which probably led the ancients to believe that the arteries (as their name implies) contained nothing but spirits during the life of an animal The true cause of the difference is this perhaps that as there is no passage to the arteries, save through the lungs and the heart, when an animal has ceased to breathe and the lungs to move, the blood in the pulmonary artery is prevented from passing into the pulmonary veins, and from thence into the left ventricle of the heart, just as we have already seen the same transit prevented in the embryo, by the want of movement in the lungs and the alternate opening and shutting of their minute orifices and invisible pores But the heart not ceasing to act at the same precise moment as the lungs, but surviving them and continuing to pulsate for a time, the left ventricle and arteries go on distributing their blood to the body at large and sending it into the veins, receiving none from the lungs, however, they are soon exhausted and left, as it were, empty But even this fact confirms our views, in no trifling manner, seeing that it can be ascribed to no other than the cause we have just assumed

Moreover it appears from this that the more frequently or forcibly the arteries pulsate, the more speedily will the body be exhausted in an hemorrhage Hence, also, it happens, that in fainting fits and in states of alarm, when the heart beats more languidly and with less force, hemorrhages are diminished or arrested

Still further, it is from this that after death, when the heart has ceased to beat, it is impossible by dividing either the jugular or femoral veins and arteries, by any effort to force out more than one-half of the whole mass of the blood Neither could the butcher, did he neglect to cut the throat of the ox which he has knocked on the head and stunned, until the heart had ceased beating, ever bleed the carcass effectually

CHAPTER X

THE FIRST POSITION* OF THE QUANTITY OF BLOOD PASSING FROM THE VEINS TO THE ARTERIES AND THAT THERE IS A CIRCUIT OF THE BLOOD, FREED FROM OBJECTIONS, AND FARTHER CONFIRMED BY EXPERIMENT

So far our first position is confirmed, whether the thing be referred to calculation or to experiment and dissection, viz, that the blood is incessantly infused into the arteries in larger quantities than it can be supplied by the food, so that the whole passing over in a short space of time, it is a matter of necessity that the blood perform a circuit, that it return to whence it set out

But if any one shall here object that a large quantity may pass through and yet no necessity be found for a circulation, that all may come from the meat and drink consumed, and quote as an illustration the abundant supply of milk in the mammae—for a cow will give three, four, and even seven gallons and more in a day, and a woman, two or three pints whilst nursing a child or twins, which must manifestly be derived from the food consumed, it may be answered, that the heart by computation does as much and more in the course of an hour or two

And if not yet convinced, he shall still insist, that when an artery is divided a preternatural route is, as it were, opened, and that so the blood escapes in torrents, but that the same thing does not happen in the healthy and uninjured body when no outlet is made, and that in arteries filled, or in their natural state, so large a quantity of blood cannot pass in so short a space of time as to make any return necessary, —to all this it may be answered, that from the calculation already made, and the reasons assigned, it appears that by so much as the heart in its dilated state contains in addition to its contents in the state of constriction, so much in a general way must it emit upon each pulsation, and in such quantity must the blood pass, the body being healthy and naturally constituted

But in serpents, and several fishes, by tying the veins some way below the heart, you will perceive a space between the ligature and the heart speedily to become empty, so that, unless you would deny the evidence of your senses, you must needs admit the return of the blood to the heart The same thing will also plainly appear when we come to discuss our second position

*Proposition —F A W 1940

Let us here conclude with a single example confirming all that has been said, and from which every one may obtain conviction through the testimony of his own eyes

If a live snake be laid open, the heart will be seen pulsating quietly, distinctly for more than an hour, moving like a worm, contracting in its longitudinal dimensions (for it is of an oblong shape) and propelling its contents, becoming of a paler colour in the systole, of a deeper tint in the diastole, and almost all things else by which I have already said that the truth I contend for is established, only that here everything takes place more slowly, and is more distinct This point in particular may be observed more clearly than the noonday sun the vena cava enters the heart at its lower part, the artery quits it at the superior part, the vein being now seized either with forceps or between the finger and thumb, and the course of the blood for some space below the heart interrupted, you will perceive the part that intervenes between the fingers and the heart almost immediately to become empty, the blood being exhausted by the action of the heart at the same time the heart will become of a much paler colour even in its state of dilatation, than it was before, it is also smaller than at first from wanting blood, and then it begins to beat more slowly, so that it seems at length as if it were about to die But the impediment to the flow of blood being removed, instantly the colour and the size of the heart are restored

If, on the contrary, the artery instead of the vein be compressed or tied, you will observe the part between the obstacle and the heart, and the heart itself to become inordinately distended, to assume a deep purple or even livid colour, and at length to be so much oppressed with blood that you will believe it about being choked, but the obstacle removed, all things immediately return to their pristine state—the heart to its colour, size, stroke, etc

Here, then, we have evidence of two kinds of death extinction from deficiency, and suffocation from excess Examples of both have now been set before you, and you have had opportunity of viewing the truth contended for with your own eyes in the heart

CHAPTER XI

THE SECOND POSITION IS DEMONSTRATED

That this may the more clearly appear to every one, I have here to cite certain experiments, from which it seems obvious that the blood enters a limb by the arteries, and returns from it by the veins, that the arteries are the vessels carrying the blood from the heart, and the veins the returning channels of the blood to the heart, that in the limbs and extreme parts of the body the blood passes either immediately by anastomosis from the arteries into the veins, or mediately by the pores of the flesh, or in both

ways, as has already been said in speaking of the passage of the blood through the lungs; whence it appears manifest that in the circuit the blood moves from thence hither and from hence thither, from the centre to the extremities, to wit, in from the extreme parts back again to the centre. Finally, upon the grounds of calculation, with the same elements as before, it will be obvious that the quantity can neither be accounted for by the ingesta, nor yet be held necessary to nutrition.

The same thing will also appear in regard to ligatures, and wherefore they are said to *draw*, though this is neither from the heat, nor the pain, nor the vacuum they occasion nor indeed from any other cause yet thought of, it will also explain the uses and advantages to be derived from ligatures in medicine, the principle upon which they either suppress or occasion hemorrhage, how they induce sloughing and more extensive mortification in extremities, and how they act in the castration of animals and the removal of warts and fleshy tumours. But it has come to pass, from no one having duly weighed and understood the causes and rationale of these various effects, that though almost all, upon the faith of the old writers recommend ligatures in the treatment of disease, yet very few comprehend their proper employment, or derive any real assistance from them in effecting cures.

Ligatures are either very tight or of middling tightness. A ligature I designate as tight or perfect when it is drawn so close about an extremity that no vessel can be felt pulsating beyond it. Such a ligature we use in amputations to control the flow of blood, and such also are employed in the castration of animals and the removal of tumours. In the latter instances, all afflux of nutriment and heat being prevented by the ligature, we see the testes and large fleshy tumours dwindle, and die, and finally fall off.

Ligatures of middling tightness I regard as those which compress a limb firmly all around, but short of pain, and in such a way as still suffers a certain degree of pulsation to be felt in the artery beyond them. Such a ligature is in use in blood letting, an operation in which the fillet applied above the elbow is not drawn so tight but that the arteries at the wrist may still be felt beating under the finger.

Now let any one make an experiment upon the arm of a man, either using such a fillet as is employed in blood letting, or grasping the limb lightly with his hand, the best subject for it being one who is lean, and who has large veins, and the best time after exercise, when the body is warm, the pulse is full, and the blood carried in larger quantity to the extremities, for all then is more conspicuous; under such circumstances let a ligature be thrown about the extremity, and drawn as tightly as can be borne, it will first be perceived that beyond the ligature, neither in the wrist nor anywhere else, do the arteries pulsate, at the same time that immediately above the ligature the artery begins to rise higher at each

diastole, to throb more violently, and to swell in its vicinity with a kind of tide, as if it strove to break through and overcome the obstacle to its current; the artery here, in short, appears as if it were preternaturally full. The hand under such circumstances retains its natural colour and appearance; in the course of time it begins to fall somewhat in temperature, indeed, but nothing is *drawn* into it.

After the bandage has been kept on for some short time in this way, let it be slackened a little, brought to that state or term of middling tightness which is used in bleeding, and it will be seen that the whole hand and arm will instantly become deeply suffused and distended, and the veins show themselves tumid and knotted; after ten or fifteen pulses of the artery, the hand will be perceived excessively distended, injected, gorged with blood, *drawn*, as it is said, by this middling ligature, without pain or heat, or any horror of a vacuum, or any other cause yet indicated.

If the finger be applied over the artery as it is pulsating by the edge of the fillet, at the moment of slackening it, the blood will be felt to glide through, as it were, underneath the finger; and he, too, upon whose arm the experiment is made, when the ligature is slackened, is distinctly conscious of a sensation of warmth, and of something, viz., a stream of blood suddenly making its way along the course of the vessels and diffusing itself through the hand, which at the same time begins to feel hot, and becomes distended.

As we had noted, in connexion with the tight ligature, that the artery above the bandage was distended and pulsated, not below it, so, in the case of the moderately tight bandage, on the contrary, do we find that the veins below, never above, the fillet, swell, and become dilated, whilst the arteries shrink; and such is the degree of distension of the veins here, that it is only very strong pressure that will force the blood beyond the fillet, and cause any of the veins in the upper part of the arm to rise.

From these facts it is easy for every careful observer to learn that the blood enters an extremity by the arteries; for when they are effectually compressed nothing is *drawn* to the member; the hand preserves its colour; nothing flows into it, neither is it distended; but when the pressure is diminished, as it is with the bleeding fillet, it is manifest that the blood is instantly thrown in with force, for then the hand begins to swell; which is as much as to say, that when the arteries pulsate the blood is flowing through them, as it is when the moderately tight ligature is applied; but where they do not pulsate, as, when a tight ligature is used, they cease from transmitting anything, they are only distended above the part where the ligature is applied. The veins again being compressed, nothing can flow through them; the certain indication of which is, that below the ligature they are much more tumid than above it, and than they usually appear when there is no bandage upon the arm.

It therefore plainly appears that the ligature prevents the return of the blood through the veins to the parts above it, and maintains those beneath it in a state of permanent distension But the arteries, in spite of its pressure, and under the force and impulse of the heart, send on the blood from the internal parts of the body to the parts beyond the bandage And herein consists the difference between the tight and the medium bandage, that the former not only prevents the passage of the blood in the veins, but in the arteries also, the latter, however, whilst it does not prevent the pulsific force from extending beyond it, and so propelling the blood to the extremities of the body, compresses the veins, and greatly or altogether impedes the return of the blood through them

Seeing, therefore, that the moderately tight ligature renders the veins turgid, and the whole hand full of blood, I ask, whence is this? Does the blood accumulate below the ligature coming through the veins, or through the arteries, or passing by certain secret pores? Through the veins it cannot come, still less can it come by any system of invisible pores, it must needs arrive by the arteries, then, in conformity with all that has been already said That it cannot flow in by the veins appears plainly enough from the fact that the blood cannot be forced towards the heart unless the ligature be removed, when on a sudden all the veins collapse, and disgorge themselves of their contents into the superior parts, the hand at the same time resuming its natural pale colour,—the tumefaction and the stagnating blood have disappeared

Moreover, he whose arm or wrist has thus been bound for some little time with the medium bandage, so that it has not only got swollen and livid but cold, when the fillet is undone is aware of something cold making its way upwards along with the returning blood, and reaching the elbow or the axilla And I have myself been inclined to think that this cold blood rising upwards to the heart was the cause of the fainting that often occurs after bloodletting fainting frequently supervenes even in robust subjects, and mostly at the moment of undoing the fillet, as the vulgar say, from the turning of the blood

Farther, when we see the veins below the ligature instantly swell up and become gorged, when from extreme tightness it is somewhat relaxed, the arteries meantime continuing unaffected, this is an obvious indication that the blood passes from the arteries into the veins, and not from the veins into the arteries, and that there is either an anastomosis of the two orders of vessels, or pores in the flesh and solid parts generally that are permeable to the blood It is farther an indication that the veins have frequent communications with one another, because they all become turgid together, whilst under the medium ligature applied above the elbow, and if any single small vein be pricked with a lancet, they all speedily shrink, and disburthening themselves into this they subside almost simultaneously

These considerations will enable any one to understand the nature of the attraction that is exerted by ligatures and perchance of fluxes generally, how, for example, the veins when compressed by a bandage of medium tightness applied above the elbow the blood cannot escape, whilst it still continues to be driven in to wit by the forcing power of the heart, by which the parts are by necessity filled, gorged with blood And how should it be otherwise? Heat and pain and the *vis vacui* draw, indeed, but in such wise only that parts are filled not preternaturally distended or gorged, not so suddenly and violently overwhelmed with the charge of blood forced in upon them that the flesh is lacerated and the vessels ruptured Nothing of the kind as an effect of heat, or pain, or the vacuum force is either credible or demonstrable

Besides, the ligature is competent to occasion the afflux in question without either pain, or heat or *vis vacui* Were pain in any way the cause how should it happen that with the arm bound above the elbow the hand and fingers should swell below the bandage, and their veins become distended? The pressure of the bandage certainly prevents the blood from getting there by the veins And then wherefore is there neither swelling nor repletion of the veins nor any sign or symptom of attraction or afflux, above the ligature? But this is the obvious cause of the preternatural attraction and swelling below the bandage and in the hand and fingers, that the blood is entering abundantly and with force but cannot pass out again

Now is not this the cause of all tumefaction as indeed Avicenna has it, and of all oppressive redundancy in parts that the access to them is open, but the egress from them is closed? Whence it comes that they are gorged and tumefied And may not the same thing happen in local inflammations, where, so long as the swelling is on the increase, and has not reached its extreme term, a full pulse is felt in the part especially when the disease is of the more acute kind and the swelling usually takes place most rapidly But these are matters for after discussion Or does this, which occurred in my own case happen from the same cause Thrown from a carriage upon one occasion, I struck my forehead a blow upon the place where a twig of the artery advances from the temple, and immediately, within the time in which twenty beats could have been made, I felt a tumour the size of an egg developed, without either heat or any great pain the near vicinity of the artery had caused the blood to be effused into the bruised part with unusual force and quickness

And now, too, we understand wherefore in phlebotomy we apply our fillet above the part that is punctured, not below it, did the flow come from above, not from below, the bandage in this case would not only be of no service, but would prove a positive hinderance, it would have to be applied below the orifice, in order to have the flow more free, did the blood descend by the veins from superior to inferior parts, but as it is elsewhere forced through the extreme arteries into the extreme veins,

and the return in these last is opposed by the ligature, so do they fill and swell, and being thus filled and distended, they are made capable of projecting their charge with force, and to a distance, when any of them is suddenly punctured, but the fillet being slackened, and the returning channels thus left open, the blood forthwith no longer escapes, save by drops, and, as all the world knows, if in performing phlebotomy the bandage be either slackened too much or the limb be bound too tightly, the blood escapes without force, because in the one case the returning channels are not adequately obstructed, in the other the channels of influx, the arteries, are impeded

CHAPTER XII

THAT THERE IS A CIRCULATION OF THE BLOOD IS SHOWN FROM THE SECOND POSITION DEMONSTRATED

If these things be so, another point which I have already referred to, viz, the continual passage of the blood through the heart, will also be confirmed We have seen, that the blood passes from the arteries into the veins, not from the veins into the arteries, we have seen, farther, that almost the whole of the blood may be withdrawn from a puncture made in one of the cutaneous veins of the arm if a bandage properly applied be used, we have seen, still farther, that the blood flows so freely and rapidly that not only is the whole quantity which was contained in the arm beyond the ligature, and before the puncture was made, discharged, but the whole which is contained in the body, both that of the arteries and that of the veins

Whence we must admit, first, that the blood is sent along with an impulse, and that it is urged with force below the fillet, for it escapes with force, which force it receives from the pulse and power of the heart, for the force and motion of the blood are derived from the heart alone Second, that the afflux proceeds from the heart, and through the heart by a course from the great veins [into the aorta], for it gets into the parts below the ligature through the arteries, not through the veins, and the arteries nowhere receive blood from the veins, nowhere receive blood save and except from the left ventricle of the heart Nor could so large a quantity of blood be drawn from one vein (a ligature having been duly applied), nor with such impetuosity, such readiness, such celerity, unless through the medium of the impelling power of the heart

But if all things be as they are now represented, we shall feel ourselves at liberty to calculate the quantity of the blood, and to reason on its circular motion Should any one, for instance, in performing phlebotomy, suffer the blood to flow in the manner it usually does, with force and freely, for some half hour or so, no question but that the greatest part of

the blood being abstracted, faintings and syncopes would ensue, and that not only would the arteries but the great veins also be nearly emptied of their contents. It is only consonant with reason to conclude that in the course of the half hour hinted at, so much as has escaped has also passed from the great veins through the heart into the aorta. And further, if we calculate how many ounces flow through one arm or how many pass in twenty or thirty pulsations under the medium ligature, we shall have some grounds for estimating how much passes through the other arm in the same space of time: how much through both lower extremities, how much through the neck on either side, and through all the other arteries and veins of the body, all of which have been supplied with fresh blood, and as this blood must have passed through the lungs and ventricles of the heart, and must have come from the great veins,—we shall perceive that a circulation is absolutely necessary, seeing that the quantities hinted at cannot be supplied immediately from the ingesta, and are vastly more than can be requisite for the mere nutrition of the parts.

It is still further to be observed, that the truths contended for are sometimes confirmed in another way; for having tied up the arm properly, and made the puncture duly, still, if from alarm or any other causes a state of faintness supervenes, in which the heart always pulsates more languidly, the blood does not flow freely, but distils by drops only. The reason is, that with the somewhat greater than usual resistance offered to the transit of the blood by the bandage, coupled with the weaker action of the heart, and its diminished impelling power, the stream cannot make its way under the fillet; and farther, owing to the weak and languishing state of the heart, the blood is not transferred in such quantity as wont from the veins to the arteries through the sinuses of that organ. So also, and for the same reasons, are the menstrual fluxes of women, and indeed hemorrhages of every kind, controlled. And now, a contrary state of things occurring, the patient getting rid of his fear and recovering his courage, the pulsific power is increased, the arteries begin again to beat with greater force, and to drive the blood even into the part that is bound, so that the blood now springs from the puncture in the vein, and flows in a continuous stream.

CHAPTER XIII

THE THIRD POSITION IS CONFIRMED: AND THE CIRCULATION OF THE BLOOD IS DEMONSTRATED FROM IT

Thus far we have spoken of the quantity of blood passing through the heart and the lungs in the centre of the body, and in like manner from the arteries into the veins in the peripheral parts and the body at large. We have yet to explain, however, in what manner the blood finds its

way back to the heart from the extremities by the veins, and how and in what way these are the only vessels that convey the blood from the external to the central parts, which done, I conceive that the three fundamental propositions laid down for the circulation of the blood will be so plain, so well established, so obviously true, that they may claim general credence Now the remaining position will be made sufficiently clear from the valves which are found in the cavities of the veins themselves, from the uses of these, and from experiments cognizable by the senses

The celebrated Hieronymus Fabricius of Aquapendente, a most skilful anatomist, and venerable old man, or, as the learned Riolan will have it, Jacobus Sylvius, first gave representations of the valves in the veins, which consist of raised or loose portions of the inner membranes of these vessels, of extreme delicacy, and a sigmoid or semilunar shape They are situated at different distances from one another, and diversely in different individuals, they are connate at the sides of the veins, they are directed upwards or towards the trunks of the veins, the two—for there are for the most part two together—regard each other, mutually touch, and are so ready to come into contact by their edges, that if anything attempt to pass from the trunks into the branches of the veins, or from the greater vessels into the less, they completely prevent it, they are farther so arranged, that the horns of those that succeed are opposite the middle of the convexity of those that precede, and so on alternately

The discoverer of these valves did not rightly understand their use, nor have succeeding anatomists added anything to our knowledge for their office is by no means explained when we are told that it is to hinder the blood, by its weight, from all flowing into inferior parts, for the edges of the valves in the jugular veins hang downwards, and are so contrived that they prevent the blood from rising upwards, the valves, in a word, do not invariably look upwards, but always towards the trunks of the veins, invariably towards the seat of the heart I, and indeed others, have sometimes found valves in the emulgent veins, and in those of the mesentery, the edges of which were directed towards the vena cava and vena portae Let it be added that there are no valves in the arteries [save at their roots], and that dogs, oxen, etc, have invariably valves at the divisions of their crural veins, in the veins that meet towards the top of the os sacrum, and in those branches which come from the haunches, in which no such effect of gravity from the erect position was to be apprehended Neither are there valves in the jugular veins for the purpose of guarding against apoplexy, as some have said, because in sleep the head is more apt to be influenced by the contents of the carotid arteries Neither are the valves present, in order that the blood may be retained in the divarications or smaller trunks and minuter branches, and not to be suffered to flow entirely into the

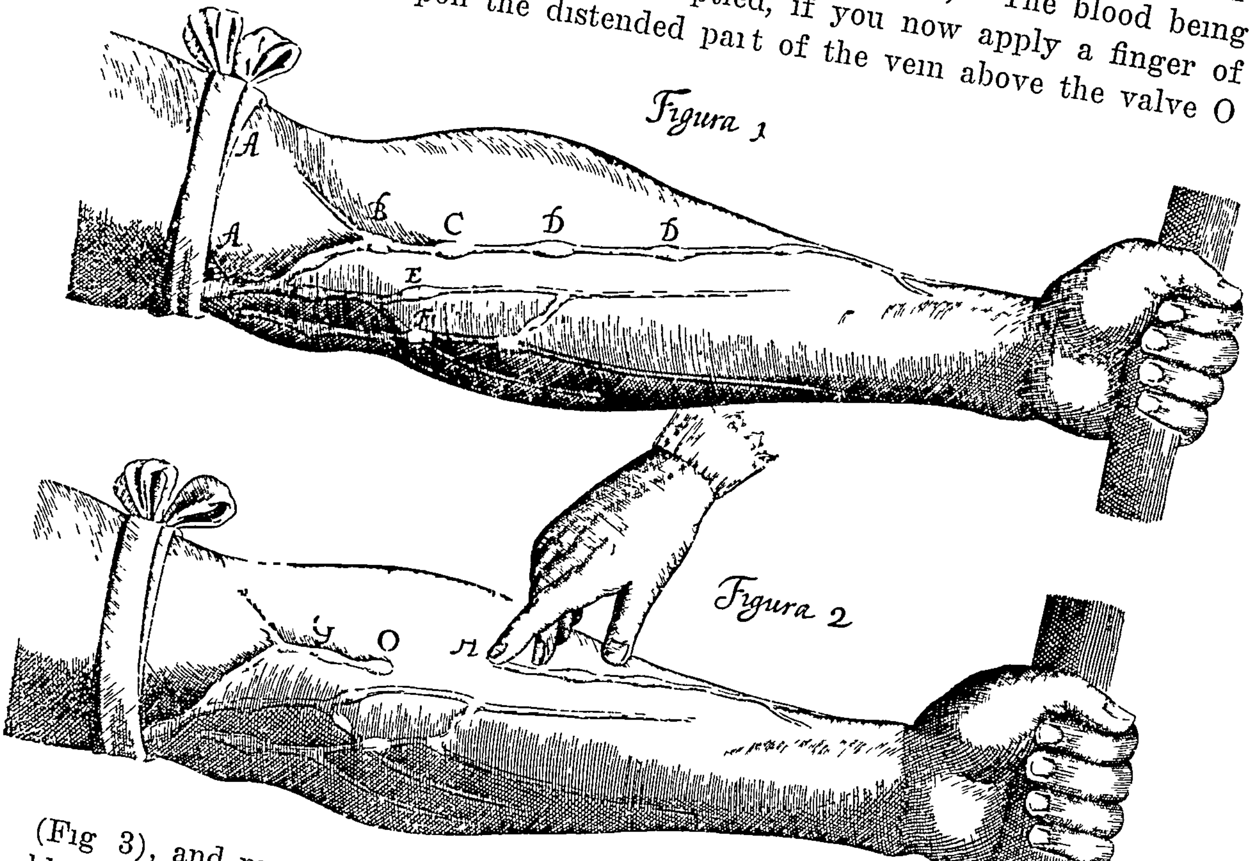
more open and capacious channels, for they occur where there are no divarications, although it must be owned that they are most frequent at the points where branches join Neither do they exist for the purpose of rendering the current of blood more slow from the centre of the body, for it seems likely that the blood would be disposed to flow with sufficient slowness of its own accord, as it would have to pass from larger into continually smaller vessels, being separated from the mass and fountain head, and attaining from warmer into colder places

But the valves are solely made and instituted lest the blood should pass from the greater into the lesser veins, and either rupture them or cause them to become varicose, lest, instead of advancing from the extreme to the central parts of the body, the blood should rather proceed along the veins from the centre to the extremities, but the delicate valves, while they readily open in the right direction, entirely prevent all such contrary motion, being so situated and arranged, that if anything escapes, or is less perfectly obstructed by the cornua of the one above the fluid passing, as it were, by the chinks between the cornua, it is immediately received on the convexity of the one beneath, which is placed transversely with reference to the former, and so is effectually hindered from getting any farther

And this I have frequently experienced in my dissections of the veins if I attempted to pass a probe from the trunk of the veins into one of the smaller branches, whatever care I took I found it impossible to introduce it far any way, by reason of the valves, whilst, on the contrary, it was most easy to push it along in the opposite direction, from without inwards, or from the branches towards the trunks and roots In many places two valves are so placed and fitted, that when raised they come exactly together in the middle of the vein, and are there united by the contact of their margins, and so accurate is the adaptation, that neither by the eye nor by any other means of examination can the slightest chink along the line of contact be perceived But if the probe be now introduced from the extreme towards the more central parts, the valves, like the floodgates of a river, give way, and are most readily pushed aside The effect of this arrangement plainly is to prevent all motion of the blood from the heart and vena cava, whether it be upwards towards the head, or downwards towards the feet, or to either side towards the arms, not a drop can pass, all motion of the blood, beginning in the larger and tending towards the smaller veins, is opposed and resisted by them, whilst the motion that proceeds from the lesser to end in the larger branches is favoured, or, at all events, a free and open passage is left for it

But that this truth may be made the more apparent, let an arm be tied up above the elbow as if for phlebotomy (A, A, Fig 1) At intervals in the course of the veins, especially in labouring people and those whose

veins are large, certain knots or elevations (B, C, D, E, F) will be perceived, and this not only at the places where a branch is received (E, F), but also where none enters (C, D) these knots or risings are all formed by valves, which thus show themselves externally And now if you press the blood from the space above one of the valves, from H to O (Fig 2), and keep the point of a finger upon the vein inferiorly, you will see no influx of blood from above, the portion of the vein between the point of the finger and the valve O will be obliterated, yet will the vessel continue sufficiently distended above that valve (O, G) The blood being thus pressed out, and the vein emptied, if you now apply a finger of the other hand upon the distended part of the vein above the valve O

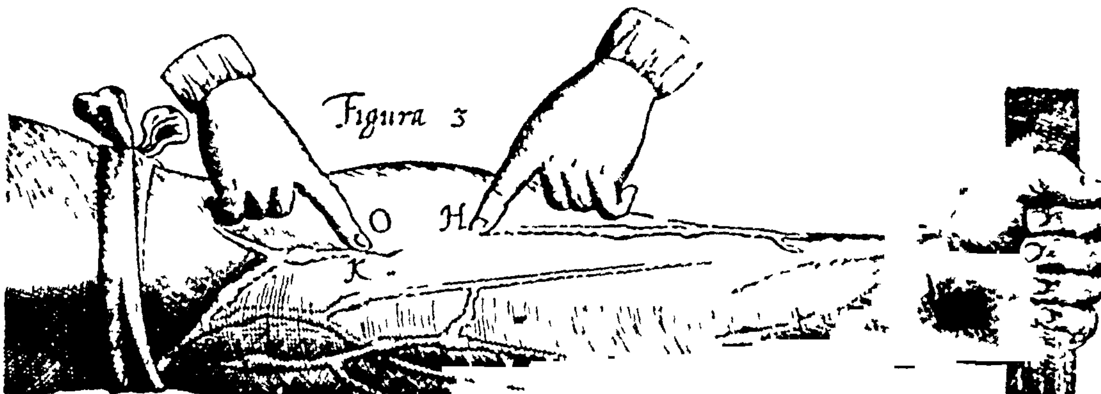


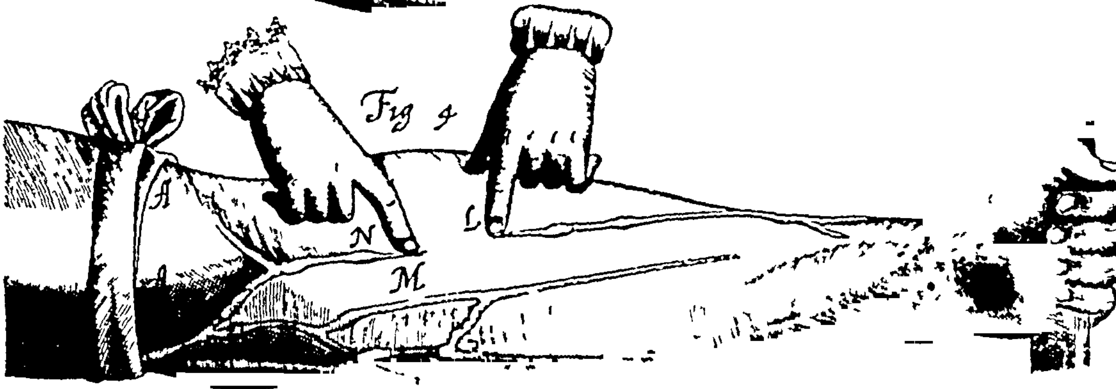
(Fig 3), and press downwards, you will find that you cannot force the blood through or beyond the valve, but the greater effort you use, you will only see the portion of vein that is between the finger and the valve become more distended, that portion of the vein which is below the valve remaining all the while empty (H, O, Fig 3)

It would therefore appear that the function of the valves in the veins is the same as that of the three sigmoid valves which we find at the commencement of the aorta and pulmonary artery, viz, to prevent all reflux of the blood that is passing over them

Farther, the arm being bound as before, and the veins looking full and distended, if you press at one part in the course of a vein with the point of a finger (L, Fig 4), and then with another finger streak the blood

upwards beyond the next valve (N), you will perceive that this portion of the vein continues empty (L N), and that the blood cannot retrograde, precisely as we have already seen the case to be in Fig 2, but the finger first applied (H, Fig 2, L, Fig 4), being removed, immediately the vein is filled from below, and the arm becomes as it appears at D C, Fig 1 That the blood in the veins therefore proceeds from inferior or more remote to superior parts, and towards the heart moving in these vessels in this and not in the contrary direction, appears most obviously And although in some places the valves, by not acting with such perfect accuracy, or where there is but a single valve do not seem totally to prevent the passage of the blood from the centre, still the greater number of them





plainly do so, and then, where things appear contrived more negligently, this is compensated either by the more frequent occurrence or more perfect action of the succeeding valves or in some other way the veins, in short, as they are the free and open conduits of the blood returning *to* the heart, so are they effectually prevented from serving as its channels of distribution *from* the heart

But this other circumstance has to be noted The arm being bound, and the veins made turgid, and the valves prominent, as before, apply the thumb or finger over a vein in the situation of one of the valves in such a way as to compress it, and prevent any blood from passing upwards from the hand, then, with a finger of the other hand, streak the

blood in the vein upwards till it has passed the next valve above, the vessel now remains empty, but the finger being removed for an instant, the vein is immediately filled from below, apply the finger again, and having in the same manner streaked the blood upwards, again remove the finger below, and again the vessel becomes distended as before, and this repeat, say a thousand times, in a short space of time And now compute the quantity of blood which you have thus pressed up beyond the valve, and then multiplying the assumed quantity by one thousand, you will find that so much blood has passed through a certain portion of the vessel, and I do now believe that you will find yourself convinced of the circulation of the blood, and of its rapid motion But if in this experiment you say that a violence is done to nature, I do not doubt but that, if you proceed in the same way, only taking as great a length of vein as possible, and merely remark with what rapidity the blood flows upwards, and fills the vessel from below, you will come to the same conclusion

CHAPTER XIV

CONCLUSION OF THE DEMONSTRATION OF THE CIRCULATION

And now I may be allowed to give in brief my view of the circulation of the blood, and to propose it for general adoption

Since all things, both argument and ocular demonstration, show that the blood passes through the lungs and heart by the action of the [auricles and] ventricles, and is sent for distribution to all parts of the body, where it makes its way into the veins and pores of the flesh, and then flows by the veins from the circumference on every side to the centre, from the lesser to the greater veins, and is by them finally discharged into the vena cava and right auricle of the heart, and this in such a quantity or in such a flux and reflux thither by the arteries, hither by the veins, as cannot possibly be supplied by the ingesta, and is much greater than can be required for mere purposes of nutrition, it is absolutely necessary to conclude that the blood in the animal body is impelled in a circle, and is in a state of ceaseless motion, that this is the act or function which the heart performs by means of its pulse, and that it is the sole and only end of the motion and contraction of the heart

CHAPTER XV

THE CIRCULATION OF THE BLOOD IS FURTHER CONFIRMED BY PROBABLE REASONS

It will not be foreign to the subject if I here show further, from certain familiar reasonings, that the circulation is matter both of convenience and necessity In the first place, since death is a corruption which takes place

through deficiency of heat[1] and since all living things are warm, all dying things cold, there must be a particular seat and fountain, a kind of home and hearth, where the cherisher of nature, the original of the native fire, is stored and preserved, whence heat and life are dispensed to all parts as from a fountain head, whence sustenance may be derived, and upon which concoction and nutrition, and all vegetative energy may depend Now, that the heart is this place, that the heart is the principle of life, and that all passes in the manner just mentioned, I trust no one will deny

The blood, therefore, required to have motion, and indeed such a motion that it should return again to the heart, for sent to the external parts of the body far from its fountain, as Aristotle says and without motion, it would become congealed For we see motion generating and keeping up heat and spirits under all circumstances, and rest allowing them to escape and be dissipated The blood, therefore, become thick or congealed by the cold of the extreme and outward parts and robbed of its spirits, just as it is in the dead, it was imperative that from its fount and origin, it should again receive heat and spirits, and all else requisite to its preservation—that, by returning, it should be renovated and restored

We frequently see how the extremities are chilled by the external cold, how the nose and cheeks and hands look blue, and how the blood, stagnating in them as in the pendent or lower parts of a corpse becomes of a dusky hue, the limbs at the same time getting torpid, so that they can scarcely be moved, and seem almost to have lost their vitality Now they can by no means be so effectually, and especially so speedily restored to heat and colour and life, as by a new afflux and appulsion of heat from its source But how can parts attract in which the heat and life are almost extinct? Or how should they whose passages are filled with condensed and frigid blood, admit fresh aliment—renovated blood—unless they had first got rid of their old contents? Unless the heart were truly that fountain where life and heat are restored to the refrigerated fluid, and whence new blood, warm, imbued with spirits, being sent out by the arteries, that which has become cooled and effete is forced on, and all the particles recover their heat which was failing, and their vital stimulus well-nigh exhausted

Hence it is that if the heart be unaffected, life and health may be restored to almost all the other parts of the body, but the heart being chilled, or smitten with any serious disease, it seems matter of necessity that the whole animal fabric should suffer and fall into decay When the source is corrupted, there is nothing, as Aristotle says,[2] which can be of service either to it or aught that depends on it And hence, by the way, it may perchance be wherefore grief, and love, and envy, and anxiety, and all affections of the mind of a similar kind are accompanied with emaciation and decay, or with cacochemy and crudity, which engender all manner of

[1]Aristotle's De Respiratione lib ii et iii De Part Animal et alibi
[2]De Part Animal iii

diseases and consume the body of man For every affection of the mind that is attended with either pain or pleasure, hope or fear, is the cause of an agitation whose influence extends to the heart, and there induces change from the natural constitution, in the temperature, the pulse and the rest, which impairing all nutrition in its source and abating the powers at large, it is no wonder that various forms of incurable disease in the extremities and in the trunk are the consequence, inasmuch as in such circumstances the whole body labours under the effects of vitiated nutrition and a want of native heat

Moreover, when we see that all animals live through food concocted in their interior, it is imperative that the digestion and distribution be perfect, and, as a consequence, that there be a place and receptacle where the aliment is perfected and whence it is distributed to the several members Now this place is the heart, for it is the only organ of the body which contains blood for the general use, all the others receive it merely for their peculiar or private advantage, just as the heart also has a supply for its own especial behoof in its coronary veins and arteries, but it is of the store which the heart contains in its auricles and ventricles that I here speak, and then the heart is the only organ which is so situated and constituted that it can distribute the blood in due proportion to the several parts of the body, the quantity sent to each being according to the dimensions of the artery which supplies it, the heart serving as a magazine or fountain ready to meet its demands

Further, a certain impulse or force, as well as an impeller or forcer, such as the heart, was required to effect this distribution and motion of the blood, both because the blood is disposed from slight causes, such as cold, alarm, horror, and the like, to collect in its source, to concentrate like parts to a whole, or the drops of water spilt upon a table to the mass of liquid, and then because it is forced from the capillary veins into the smaller ramifications, and from these into the larger trunks by the motion of the extremities and the compression of the muscles generally The blood is thus more disposed to move from the circumference to the centre than in the opposite direction, were there even no valves to oppose its motion, whence that it may leave its source and enter more confined and colder channels, and flow against the direction to which it spontaneously inclines, the blood requires both force and an impelling power Now such is the heart and the heart alone, and that in the way and manner already explained

CHAPTER XVI

THE CIRCULATION OF THE BLOOD IS FURTHER PROVED FROM CERTAIN CONSEQUENCES

There are still certain phenomena, which, taken as consequences of this truth assumed as proven, are not without their use in exciting belief, as it were, *a posteriore*; and which, although they may seem to be involved in much doubt and obscurity, nevertheless readily admit of having reasons and causes assigned for them. The phenomena alluded to are those that present themselves in connexion with contagions, poisoned wounds, the bites of serpents and rabid animals, lues venerea and the like. We sometimes see the whole system contaminated, though the part first infected remains sound; the lues venerea has occasionally made its attack with pains in the shoulders and head, and other symptoms, the genital organs being all the while unaffected; and then we know that the wound made by the rabid dog having healed, fever and a train of disastrous symptoms nevertheless supervene. Whence it appears that the contagion impressed upon or deposited in a particular part, is by and by carried by the returning current of blood to the heart, and by that organ is sent to contaminate the whole body.

In tertian fever, the morbific cause seeking the heart in the first instance, and hanging about the heart and lungs, renders the patient shortwinded, disposed to sighing, indisposed to exertion, because the vital principle is oppressed and the blood forced into the lungs and rendered thick, does not pass through their substance (as I have myself seen in opening the bodies of those who had died in the beginning of the attack), when the pulse is always frequent, small, and occasionally irregular; but the heat increasing, the matter becoming attenuated, the passages forced, and the transit made, the whole body begins to rise in temperature, and the pulse becomes fuller, stronger—the febrile paroxysm is fully formed, whilst the preternatural heat kindled in the heart, is thence diffused by the arteries through the whole body along with the morbific matter, which is in this way overcome and dissolved by nature.

When we perceive, further, that medicines applied externally exert their influence on the body just as if they had been taken internally, the truth we are contending for is confirmed. Colocynth and aloes [applied externally] move the belly, cantharides excites the urine, garlic applied to the soles of the feet assists expectoration, cordials strengthen, and an infinite number of examples of the same kind might be cited. It will not, therefore, be found unreasonable perchance, if we say that the veins, by means of their orifices, absorb some of the things that are applied externally and carry this inwards with the blood, not otherwise, it may be, than those of the mesentery imbibe the chyle from the intestines and carry it mixed with the blood to the liver. For the blood entering the mesentery by the

coeliac artery, and the superior and inferior mesenterics, proceeds to the intestines, from which, along with the chyle that has been attracted into the veins, it returns by their numerous ramifications into the vena portae of the liver, and from this into the vena cava, and this in such wise that the blood in these veins has the same colour and consistency as in other veins, in opposition to what many believe to be the fact. Nor indeed can we imagine two contrary motions in any capillary system—the chyle upwards, the blood downwards. This could scarcely take place, and must be held as altogether improbable. But is not the thing rather arranged as it is by the consummate providence of nature? For were the chyle mingled with the blood, the crude with the concocted, in equal proportions, the result would not be concoction, transmutation, and sanguification, but rather, and because they are severally active and passive, a mixture or combination, or medium compound of the two, precisely as happens when wine is mixed with water and syrup. But when a very minute quantity of chyle is mingled with a very large quantity of circulating blood, a quantity of chyle that bears no kind of proportion to the mass of blood, the effect is the same, as Aristotle says, as when a drop of water is added to a cask of wine, or the contrary; the mass does not then present itself as a mixture, but is still sensibly either wine or water. So in the mesenteric veins of an animal we do not find either chyme or chyle and blood, blended together or distinct, but only blood, the same in colour, consistency, and other sensible properties, as it appears in the veins generally. Still as there is a certain though small and inappreciable proportion of chyle or unconcocted matter mingled with this blood, nature has interposed the liver, in whose meandering channels it suffers delay and undergoes additional change, lest arriving prematurely and crude at the heart, it should oppress the vital principle. Hence in the embryo, there is almost no use for the liver, but the umbilical vein passes directly through, a foramen or anastomosis existing from the vena portae, so that the blood returns from the intestines of the foetus, not through the liver, but into the umbilical vein mentioned, and flows at once into the heart, mingled with the natural blood which is returning from the placenta; whence also it is that in the development of the foetus the liver is one of the organs that is last formed; I have observed all the members perfectly marked out in the human foetus, even the genital organs, whilst there was yet scarcely any trace of the liver. And indeed at the period when all the parts, like the heart itself in the beginning, are still white, and save in the veins there is no appearance of redness, you shall see nothing in the seat of the liver but a shapeless collection, as it were, of extravasated blood, which you might take for the effects of a contusion or ruptured vein.

But in the incubated egg there are, as it were, two umbilical vessels, one from the albumen passing entire through the liver, and going

straight to the heart, another from the yelk, ending in the vena portae, for it appears that the chick, in the first instance, is entirely formed and nourished by the white, but by the yelk after it has come to perfection and is excluded from the shell, for this part may still be found in the abdomen of the check many days after its exclusion, and is a substitute for the milk to other animals.

But these matters will be better spoken of in my observations on the formation of the foetus, where many propositions, the following among the number, will be discussed. Wherefore is this part formed or perfected first, that last?—and of the several members: what part is the cause of another? And many points having special reference to the heart, such as: Wherefore does it first acquire consistency, and appear to possess life, motion, sense, before any other part of the body is perfected? as Aristotle says in his third book, *De Partibus Animalium*. And so also of the blood: Wherefore does it precede all the rest? And in what way does it possess the vital and animal principle? And show a tendency to motion, and to be impelled hither and thither, the end for which the heart appears to be made? In the same way, in considering the pulse: Wherefore one kind of pulse should indicate death, another recovery? And so of all the other kinds of pulse, what may be the cause and indication of each. So also in the consideration of crises and natural critical discharges, of nutrition, and especially the distribution of the nutriment, and of defluxions of every description. Finally, reflecting on every part of medicine, physiology, pathology, semeiotics, therapeutics, when I see how many questions can be answered, how many doubts resolved, how much obscurity illustrated, by the truth we have declared, the light we have made to shine, I see a field of such vast extent in which I might proceed so far, and expatiate so widely, that this my tractate would not only swell out into a volume, which was beyond my purpose, but my whole life, perchance, would not suffice for its completion.

In this place, therefore, and that indeed in a single chapter, I shall only endeavour to refer the various particulars that present themselves in the dissection of the heart and arteries to their several uses and causes, for so I shall meet with many things which receive light from the truth I have been contending for, and which, in their turn, render it more obvious. And indeed I would have it confirmed and illustrated by anatomical arguments above all others.

There is but a single point which indeed would be more correctly placed among our observations on the use of the spleen, but which it will not be altogether impertinent to notice in this place incidentally. From the splenic branch which passes into the pancreas, and from the upper part, arise the posterior coronary, gastric, and gastroepiploic veins, all of which are distributed upon the stomach in numerous branches and twigs, just as the mesenteric vessels are upon the intestines, in like manner, from the

inferior part of the same splenic branch, and along the back of the colon and rectum proceed the hemorrhoidal veins The blood returning by these veins, and bringing the cruder juices along with it, on the one hand from the stomach, where they are thin, watery, and not yet perfectly chylified, on the other thick and more earthy, as derived from the faeces, but all poured into this splenic branch, are duly tempered by the admixture of contraries, and nature mingling together these two kinds of juices, difficult of coction by reason of most opposite defects, and then diluting them with a large quantity of warm blood (for we see that the quantity returned from the spleen must be very large when we contemplate the size of its arteries), they are brought to the porta of the liver in a state of higher preparation, the defects of either extreme are supplied and compensated by this arrangement of the veins

CHAPTER XVII

THE MOTION AND CIRCULATION OF THE BLOOD ARE CONFIRMED FROM THE PARTICULARS APPARENT IN THE STRUCTURE OF THE HEART, AND FROM THOSE THINGS WHICH DISSECTION UNFOLDS

I do not find the heart as a distinct and separate part in all animals, some indeed, such as the zoophytes, have no heart, this is because these animals are coldest, of no great bulk, of soft texture or of a certain uniform sameness or simplicity of structure, among the number I may instance grubs and earthworms, and those that are engendered of putrefaction and do not preserve their species These have no heart, as not requiring any impeller of nourishment into the extreme parts, for they have bodies which are connate and homogeneous, and without limbs, so that by the contraction and relaxation of the whole body they assume and expel, move and remove the aliment Oysters, mussels, sponges, and the whole genus of zoophytes or plant-animals have no heart, for the whole body is used as a heart, or the whole animal is a heart In a great number of animals, almost the whole tribe of insects, we cannot see distinctly by reason of the smallness of the body, still in bees, flies, hornets, and the like, we can perceive something pulsating with the help of a magnifying glass, in pediculi, also, the same thing may be seen, and as the body is transparent, the passage of food through the intestines, like a black spot or stain, may be perceived by the aid of the same magnifying glass

In some of the bloodless[1] and colder animals, further, as in snails, whelks, shrimps, and shell-fish, there is a part which pulsates—a kind of vesicle or auricle without a heart—slowly indeed, and not to be perceived save in the warmer season of the year In these creatures this part is so

[1] I e, not having red blood [Willis 1847]

contrived that it shall pulsate, as there is here a necessity for some impulse to distribute the nutritive fluid by reason of the variety of organic parts, or of the density of the substance, but the pulsations occur infrequently, and sometimes in consequence of the cold not at all, an arrangement the best adapted to them as being of a doubtful nature, so that sometimes they appear to live sometimes to die, sometimes they show the vitality of an animal, sometimes of a vegetable This seems also to be the case with the insects which conceal themselves in winter, and lie, as it were, defunct, or merely manifesting a kind of vegetative existence But whether the same thing happens in the case of certain animals that have red blood, such as frogs, tortoises, serpents, swallows, may be made a question without any kind of impropriety

In all the larger and warmer, because [red-] blooded animals, there was need of an impeller of the nutritive fluid, and that perchance possessing a considerable amount of power In fishes, serpents, lizards, tortoises, frogs, and others of the same kind there is a heart present, furnished with both an auricle and a ventricle, whence it is perfectly true, as Aristotle has observed,[1] that no [red-] blooded animal is without a heart, by the impelling power of which the nutritive fluid is forced, both with greater vigour and rapidity to a greater distance it is not merely agitated by an auricle as it is in lower forms And then in regard to animals that are yet larger, warmer, and more perfect, as they abound in blood, which is ever hotter and more spirituous, and possess bodies of greater size and consistency, they require a larger, stronger, and more fleshy heart, in order that the nutritive fluid may be propelled with yet greater force and celerity And further, inasmuch as the more perfect animals require a still more perfect nutrition, and a larger supply of native heat, in order that the aliment may be thoroughly concocted and acquire the last degree of perfection, they required both lungs and a second ventricle, which should force the nutritive fluid through them

Every animal that has lungs has therefore two ventricles to its heart, one right, and another left, and wherever there is a right, also is there a left ventricle, but the contrary of this does not hold good where there is a left there is not always a right ventricle The left ventricle I call that which is distinct in office, not in place from the other, that one namely which distributes the blood to the body at large, not to the lungs only Hence the left ventricle seems to form the principal part of the heart, situated in the middle, more strongly marked, and constructed with greater care, the heart seems formed for the sake of the left ventricle, and the right but to minister to it, for the right neither reaches to the apex of the heart, nor is it nearly of such strength, being three times thinner in its walls, and in some sort jointed on to the left, (as Aristotle says,) though

[1] De Part Animal, lib iii

indeed it is of greater capacity, inasmuch as it has not only to supply material to the left ventricle, but likewise to furnish aliment to the lungs

It is to be observed, however, that all this is otherwise in the embryo, where there is not such a difference between the two ventricles, but as in a double nut, they are nearly equal in all respects, the apex of the right reaching to the apex of the left, so that the heart presents itself as a sort of double-pointed cone And this is so, because in the foetus, as already said, whilst the blood is not passing through the lungs from the right to the left cavities of the heart, but flowing by the foramen ovale and ductus arteriosus, directly from the vena cava into the aorta, whence it is distributed to the whole body, both ventricles have in fact the same office to perform, whence their equality of constitution It is only when the lungs come to be used, and it is requisite that the passages indicated should be blocked up, that the difference in point of strength and other things between the two ventricles begins to be apparent in the altered circumstances the right has only to throw the blood through the lungs, whilst the left has to impel it through the whole body

There are further within the heart numerous braces, so to speak, fleshy columns and fibrous bands, which Aristotle, in his third book on Respiration, and the Parts of Animals, entitles nerves These are variously extended, and are either distinct or contained in grooves in the walls and partition, where they occasion numerous pits or depressions They constitute a kind of small muscles, which are superadded and supplementary to the heart, assisting it to execute a more powerful and perfect contraction, and so proving subservient to the complete expulsion of the blood They are in some sort like the elaborate and artful arrangement of ropes in a ship, bracing the heart on every side as it contracts, and so enabling it more effectually and forcibly to expel the charge of blood from its ventricles This much is plain, at all events, that some animals have them strongly marked, others have them less so, and, in all that have them, they are more numerous and stronger in the left than in the right ventricle, and whilst some have them in the left, there are yet none present in the right ventricle In the human subject, again, these fleshy columns and braces are more numerous in the left than in the right ventricle, and they are more abundant in the ventricles than in the auricles, occasionally, indeed, in the auricles there appear to be none present whatsoever In large, more muscular and hardier bodies, as of countrymen, they are numerous, in more slender frames and in females they are fewer

In those animals in which the ventricles of the heart are smooth within, and entirely without fibres or muscular bands, or anything like foveae, as in almost all the smaller birds, the partridge and the common fowl, serpents, frogs, tortoises, and also fishes, for the major part, there are no chordae tendinae, nor bundles of fibres, neither are there any tricuspid valves in the ventricles

Some animals have the right ventricle smooth internally, but the left provided with fibrous bands, such as the goose, swan, and larger birds, and the reason here is still the same as elsewhere as the lungs are spongy and loose, and soft, no great amount of force is required to force the blood through them, hence the right ventricle is either without the bundles in question, or they are fewer and weaker, not so fleshy or like muscles, those of the left ventricle, however, are both stronger and more numerous, more fleshy and muscular, because the left ventricle requires to be stronger, inasmuch as the blood which it propels has to be driven through the whole body And this, too, is the reason why the left ventricle occupies the middle of the heart, and has parietes three times thicker and stronger than those of the right Hence all animals—and among men it is not otherwise—that are endowed with particularly strong frames, and that have *large and fleshy limbs at a great distance from the heart, have this central* organ of greater thickness, strength, and muscularity And this is both obvious and necessary Those, on the contrary, that are of softer and more slender make have the heart more flaccid, softer, and internally either sparely or not at all fibrous Consider farther the use of the several valves, which are all so arranged, that the blood once received into the ventricles of the heart shall never regurgitate, once forced into the pulmonary artery and aorta shall not flow back upon the ventricles When the valves are raised and brought together they form a three cornered line, such as is left by the bite of a leech, and the more they are forced, the more firmly do they oppose the passage of blood The tricuspid valves are placed, like gate-keepers, at the entrance into the ventricles from the venae cavae and pulmonary veins, lest the blood when most forcibly impelled should flow back, and it is for this reason that they are not found in all animals, neither do they appear to have been constructed with equal care in all the animals in which they are found, in some they are more accurately fitted, in others more remissly or carelessly contrived and always with a view to their being closed under a greater or a slighter force of the ventricle In the left ventricle, therefore, and in order that the occlusion may be the more perfect against the greater impulse, there are only two valves, like a mitre, and produced into an elongated cone, so that they come together and touch to their middle, a circumstance which perhaps led Aristotle into the error of supposing this ventricle to be double, the division taking place transversely For the same reason, indeed, and that the blood may not regurgitate upon the pulmonary veins, and thus the force of the ventricle in propelling the blood through the system at large come to be neutralized, it is that these mitral valves excel those of the right ventricle in size and strength, and exactness of closing Hence, too, it is essential that there can be no heart without a ventricle, since this must be the source and storehouse of the blood The same law does not hold good in reference to the brain For almost no genus of birds has a ventricle in the

brain, as is obvious in the goose and swan, the brains of which nearly equal that of a rabbit in size; now rabbits have ventricles in the brain, whilst the goose has none. In like manner, wherever the heart has a single ventricle, there is an auricle appended, flaccid, membranous, hollow, filled with blood; and where there are two ventricles, there are likewise two auricles. On the other hand, however, some animals have an auricle without any ventricle; or at all events they have a sac analogous to an auricle; or the vein itself dilated at a particular part, performs pulsations, as is seen in hornets, bees, and other insects, which certain experiments of my own enable me to demonstrate have not only a pulse, but a respiration in that part which is called the tail, whence it is that this part is elongated and contracted now more rarely, now more frequently, as the creature appears to be blown and to require a larger quantity of air. But of these things, more in our Treatise on Respiration.

It is in like manner evident that the auricles pulsate, contract, as I have said before, and throw the blood into the ventricles; so that wherever there is a ventricle an auricle is necessary, not merely that it may serve, according to the general belief, as a source and magazine for the blood: for what were the use of its pulsations had it nothing to do save to contain? No; the auricles are prime movers of the blood, especially the right auricle, which is "the first to live, the last to die," as already said; whence they are subservient to sending the blood into the ventricle, which, contracting incontinently, more readily and forcibly expels the blood already in motion; just as the ball-player can strike the ball more forcibly and further if he takes it on the rebound than if he simply threw it. Moreover, and contrary to the general opinion, since neither the heart nor anything else can dilate or distend itself so as to draw aught into its cavity during the diastole, unless, like a sponge, it has been first compressed, and as it is returning to its primary condition; but in animals all local motion proceeds from, and has its original in the contraction of some part: it is consequently by the contraction of the auricles that the blood is thrown into the ventricles, as I have already shown, and from thence, by the contraction of the ventricles, it is propelled and distributed. Which truth concerning local motions, and how the immediate moving organ in every motion of an animal primarily endowed with a motive spirit (as Aristotle has it[1]) is contractile; and how Aristotle was acquainted with the muscles, and did not unadvisedly refer all motion in animals to the nerves, or to the contractile element, and therefore called those little bands in the heart nerves—all this, if I am permitted to proceed in my purpose of making a particular demonstration of the organs of motion in animals from observations in my possession, I trust I shall be able to make sufficiently plain.

But that we may go on with the subject we have in hand, viz., the use of the auricles in filling the ventricles: we should expect that the more

[1]In the book De Spiritu, and elsewhere.

dense and compact the heart the thicker its parietes, the stronger and more muscular must be the auricle to force and fill it, and vice versa. Now this is actually so: in some the auricle presents itself as a sanguinolent vesicle, as a thin membrane containing blood, as in fishes, in which the sac that stands in lieu of the auricle, is of such delicacy and ample capacity, that it seems to be suspended or to float above the heart; in those fishes in which the sac is somewhat more fleshy, as in the carp, barbel, tench, and others, it bears a wonderful and strong resemblance to the lungs.

In some men of sturdier frame and stouter make the right auricle is so strong, and so curiously constructed within of bands and variously interlacing fibres, that it seems to equal the ventricle of the heart in other subjects; and I must say that I am astonished to find such diversity in this particular in different individuals. It is to be observed, however, that in the foetus the auricles are out of all proportion large, which is because they are present before the heart [the ventricular portion] makes its appearance or suffices for its office even when it has appeared, and they therefore have, as it were, the duty of the whole heart committed to them, as has already been demonstrated. But what I have observed in the formation of the foetus as before remarked (and Aristotle had already confirmed all in studying the incubated egg) throws the greatest light and likelihood upon the point. Whilst the foetus is yet in the guise of a soft worm, or, as is commonly said, in the milk, there is a mere bloody point or pulsating vesicle, a portion apparently of the umbilical vein, dilated at its commencement or base; by and by, when the outline of the foetus is distinctly indicated, and it begins to have greater bodily consistence, the vesicle in question having become more fleshy and stronger, and changed its position, passes into the auricles, over or upon which the body of the heart begins to sprout, though as yet it apparently performs no duty; but when the foetus is farther advanced, when the bones can be distinguished from the soft parts, and movements take place, then it has also a heart internally which pulsates, and, as I have said, throws blood by either ventricle from the vena cava into the arteries.

Thus nature, ever perfect and divine, doing nothing in vain, has neither given a heart where it was not required, nor produced it before its office had become necessary; but by the same stages in the development of every animal, passing through the constitutions of all, as I may say (ovum, worm, foetus), it acquires perfection in each. These points will be found elsewhere confirmed by numerous observations on the formation of the foetus.

Finally, it was not without good grounds that Hippocrates, in his book, *De Corde*, intitles it as a muscle; as its action is the same, so is its function, viz., to contract and move something else, in this case, the charge of the blood.

Farther, as in muscles at large, so can we infer the action and use of the heart from the arrangement of its fibres and its general structure. All

anatomists admit with Galen that the body of the heart is made up of various courses of fibres running straight, obliquely, and transversely, with reference to one another; but in a heart which has been boiled the arrangement of the fibres is seen to be different: all the fibres in the parietes and septum are circular, as in the sphincters; those, again, which are in the columnae extend lengthwise, and are oblique longitudinally; and so it comes to pass, that when all the fibres contract simultaneously, the apex of the cone is pulled towards its base by the columnae, the walls are drawn circularly together into a globe, the whole heart in short is contracted, and the ventricles narrowed; it is therefore impossible not to perceive that, as the action of the organ is so plainly contraction, its function is to propel the blood into the arteries.

Nor are we the less to agree with Aristotle in regard to the sovereignty of the heart; nor are we to inquire whether it receives sense and motion from the brain? whether blood from the liver? whether it be the origin of the veins and of the blood? and more of the same description. They who affirm these propositions against Aristotle, overlook, or do not rightly understand the principal argument, to the effect that the heart is the first part which exists, and that it contains within itself blood, life, sensation, motion, before either the brain or the liver were in being, or had appeared distinctly, or, at all events, before they could perform any function. The heart, ready furnished with its proper organs of motion, like a kind of internal creature, is of a date anterior to the body: first formed, nature willed that it should afterwards fashion, nourish, preserve, complete the entire animal, as its work and dwelling place: the heart, like the prince in a kingdom, in whose hands lie the chief and highest authority, rules over all; it is the original and foundation from which all power is derived, on which all power depends in the animal body.

And many things having reference to the arteries farther illustrate and confirm this truth. Why does not the arteria venosa pulsate, seeing that it is numbered among the arteries? Or wherefore is there a pulse in the vena arteriosa? Because the pulse of the arteries is derived from the impulse of the blood. Why does an artery differ so much from a vein in the thickness and strength of its coats? Because it sustains the shock of the impelling heart and streaming blood. Hence, as perfect nature does nothing in vain, and suffices under all circumstances, we find that the nearer the arteries are to the heart, the more do they differ from the veins in structure; here they are both stronger and more ligamentous, whilst in extreme parts of the body, such as the feet and hands, the brain, the mesentery, and the testicles, the two orders of vessels are so much alike that it is impossible to distinguish between them with the eye. Now this is for the following very sufficient reasons: for the more remote vessels are from the heart, with so much the less force are they impinged upon by the stroke of the

heart, which is broken by the great distance at which it is given Add to this, that the impulse of the heart exerted upon the mass of blood, which must needs fill the trunks and branches of the arteries, is diverted, divided, as it were, and diminished at every subdivision, so that the ultimate capillary divisions of the arteries look like veins, and this not merely in constitution but in function, for they have either no perceptible pulse, or they rarely exhibit one, and never save where the heart beats more violently than wont, or at a part where the minute vessel is more dilated or open than elsewhere Hence it happens that at times we are aware of a pulse in the teeth, in inflammatory tumours, and in the fingers, at another time we feel nothing of the sort Hence too, by this single symptom I have ascertained for certain that young persons, whose pulses are naturally rapid, were labouring under fever, in like manner, on compressing the fingers in youthful and delicate subjects during a febrile paroxysm, I have readily perceived the pulse there On the other hand, when the heart pulsates more languidly, it is often impossible to feel the pulse not merely in the fingers, but at the wrist, and even at the temple, this is the case in persons afflicted with lipothymiae and asphyxia, and hysterical symptoms, as also in persons of very weak constitution and in the moribund

And here surgeons are to be advised that, when the blood escapes with force in the amputation of limbs, in the removal of tumors, and in wounds, it constantly comes from an artery, not always per saltum, however, because the smaller arteries do not pulsate especially if a tourniquet has been applied

And then the reason is the same wherefore the pulmonary artery has not only the structure of an artery, but wherefore it does not differ so widely in the thickness of its tunics from the veins as the aorta the aorta sustains a more powerful shock from the left ventricle than the pulmonary artery does from the right, and the tunics of this last vessel are thinner and softer than those of the aorta in the same proportion as the walls of the right ventricle of the heart are weaker and thinner than those of the left ventricle, and in like manner, in the same degree in which the lungs are softer and laxer in structure than the flesh and other constituents of the body at large, do the tunics of the branches of the pulmonary artery differ from the tunics of the vessels derived from the aorta And the same proportion in these several particulars is universally preserved The more muscular and powerful men are, the firmer their flesh, the stronger, thicker, denser, and more fibrous their heart, in the same proportion are the auricles and arteries in all respects thicker, closer, and stronger And again, and on the other hand, in those animals the ventricles of whose heart are smooth within, without villi or valves, and the walls of which are thinner, as in fishes, serpents, birds, and very many genera of animals, in all of them the arteries differ little or nothing in the thickness of their coats from the veins

Farther, the reason why the lungs have such ample vessels, both arteries and veins (for the capacity of the pulmonary veins exceeds that of both the crural and jugular vessels), and why they contain so large a quantity of blood, as by experience and ocular inspection we know they do, admonished of the fact indeed by Aristotle, and not led into error by the appearances found in animals which have been bled to death,—is, because the blood has its fountain and storehouse and the workshop of its last perfection in the heart and lungs. Why, in the same way we find in the course of our anatomical dissections the arteria venosa and left ventricle so full of blood, of the same black colour and clotted character too, as that with which the right ventricle and pulmonary artery are filled inasmuch as the blood is incessantly passing from one side of the heart to the other through the lungs. Wherefore, in fine, the pulmonary artery or vena arteriosa has the constitution of an artery, the pulmonary veins or arteriae venosae have the structure of veins, because, in sooth, in function and constitution, and everything else, the first is an artery, the others are veins, in opposition to what is commonly believed, and why the pulmonary artery has so large an orifice, because it transports much more blood than is requisite for the nutrition of the lungs.

All these appearances, and many others, to be noted in the course of dissection, if rightly weighed, seem clearly to illustrate and fully to confirm the truth contended for throughout these pages, and at the same time to stand in opposition to the vulgar opinion, for it would be very difficult to explain in any other way to what purpose all is constructed and arranged as we have seen it to be.

1640

PIERRE GASSENDI

A BRIEF NOTE UPON A DEMONSTRATION OF THE EXISTENCE OF THE FORAMEN OVALE IN THE ADULT

PIERRE GASSENDI

(Courtesy Zeitschrift für die gesamte Naturwissenschaft)

PIERRE GASSENDI

(1592-1655)

PIERRE GASSENDI was born at Champtercier, near Digne, in Provence, France His uncle, the curé of Champtercier, noticed that Gassendi had a remarkable ability for learning, and through his efforts Gassendi was sent to the college at Digne When he was nineteen years of age, Gassendi matriculated at the University at Aix, where he studied philosophy under Fesaye A year later (1612) he was recalled to the college at Digne to lecture on theology In 1616 he received the degree of Doctor of Theology at Avignon and in the following year he was ordained Following the acceptance of holy orders, he was invited to the University at Aix to occupy the chair of philosophy, which he accepted

At Aix he became interested in anatomy, physics, and astronomy, and he once remarked that he frequently observed dissections in the anatomic amphitheater at Aix He became more and more critical of the pedagogic reliance on the orthodox Aristotelio-scholastic teachings, and for this reason, presumably, he was asked to leave Aix in 1622 He next became a canon at Grenoble In 1624 at Grenoble was published his "Exercitationes paradoxicae adversus Aristoteleos" This quarto volume contained his several arguments against the teachings of Aristotle

In 1625, Gassendi was appointed to be provost of the Cathedral of Digne, but the appointment was not confirmed until 1634 During the interim he traveled first to Paris and later to Flanders and Holland During these years he published attacks against Robert Fludd, the English physician who expounded mystical philosophy From 1641 to 1646 in a series of writings he quarreled with Descartes regarding his metaphysical conceptions

Through the influence of Richelieu, Gassendi was appointed by Louis XIV to the chair of mathematics in the Collège Royal While he was in Paris, Gassendi became interested in Epicurean philosophy and later published three works on this subject

Gassendi suffered from a disease of the thorax and in 1648 left Paris to seek a milder climate in southern France In 1653, he returned to Paris but his health did not improve and after interminable bleedings by his physicians he died on October 24, 1655

In 1640, Gassendi had published, in a volume (by himself and three other authors) entitled "De foetus formatione," a brief note on a demonstration of the existence of the vestigial foramen ovale in the adult This was not Gassendi's discovery but merely a note or report of an anatomic dissection he had witnessed during his professorship at Aix It is, however, an observation of great moment, and because the question of the perviousness of the septum of the heart was an important anatomic consideration vitally necessary to the establishment of the fact of the circulation of the blood, we have chosen to reproduce it

A NICE OBSERVATION OF THE PERVIOUSNESS OF THE SEPTUM OF THE HEART

By

PETER GASSENDI

I SHALL describe what I myself have seen.

While I was residing in Aix, whenever a dissection was being performed I was present frequently in the anatomical amphitheatre. Now for many years I had observed invariably that dissectors, taking the heart in their hands, would test the perviousness of its septum with a blunt instrument which they call a spatula, and would conclude, as physicians have concluded, that the transmission of blood from the right chamber to the left must occur by insensible transudation.

Now when this problem came to be discussed by the professors of anatomy, eight years ago, there came among the disputants a diligent surgeon, Payanus by name, who wanted to demonstrate to us onlookers that the facts were otherwise. So, taking up the spatula, he undertook to penetrate the mediastinum of the heart. But he did not attempt to push the instrument straight through, as the others had done, but having introduced its tip (for the tissue of the septum presents a thousand little openings) pushed onward with utmost gentleness, turning the instrument with the greatest patience up and down and from side to side, seeking always a farther ingress. And at last the instrument was seen entering the left chamber. But then, because we alleged that he had made an artificial opening, he himself requested one of us to incise the septum down to his instrument, with a sharp scalpel. When the incision had been made we found that no tissue anywhere had been injured, and we saw that only the meatus, or canal, notwithstanding the fact that it was a very winding passage, was lined with a very thin and glistening membrane.

These, then, are the passages which I mentioned to Fludd (who acknowledged clearly that he had been ignorant of their existence too), and I said that since they really do exist they cannot be without some function, and therefore it should be evident that there is a real percolation of blood from the right chamber into the left. And I maintained that the arterial blood was derived in this way.

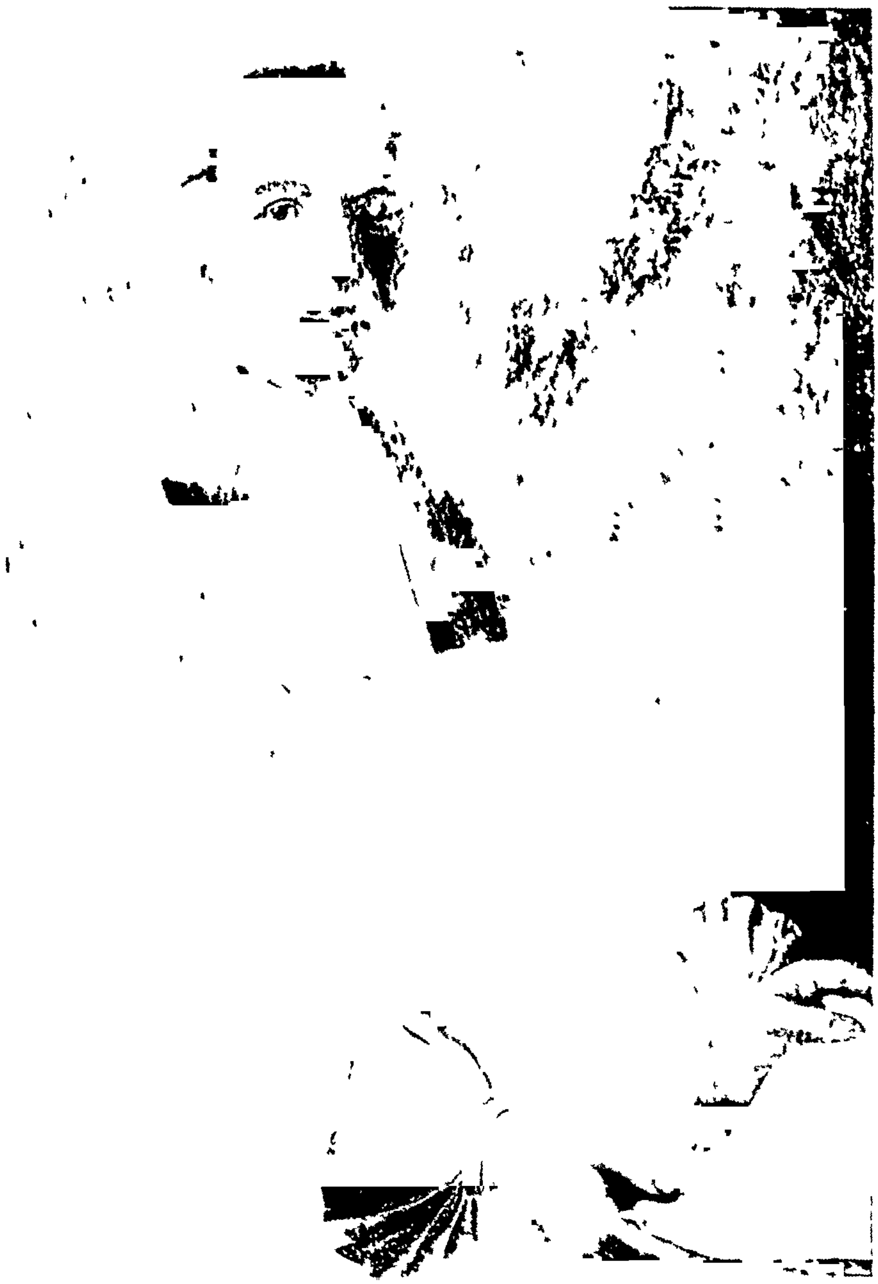
**Elegans de septo cordis pervio observatio*, 1640. Translated by G. Kasten Tallmadge. Bull. Hist. Med. 7: 429-457, 1939.

Indeed, it seems probable that the more subtle part of the blood is, so to speak, sucked through this septum, or forced through by compression But the grosser part of the blood, with the heavy vapours which it contains, enters the patent pulmonary artery and pervades and nourishes the tissues of the lungs Then, after expiration has carried off the heavy breath and the heavy vapours, the more subtle residue of this blood is gathered into the pulmonary vein so that, together with the purer air which was inhaled in breathing, it may flow into the left ventricle, either drop by drop, as the general notion says it must, or in large spurts, as Harvey's opinion seems to have it

1661

MARCELLO MALPIGHI

ON THE CAPILLARIES



MARCELLO MALPIGHI

Portrait by unknown artist

(Courtesy Charles C Thomas)

MARCELLO MALPIGHI

(1628 1694)

"I see with my eyes, a great, certain thing"

—Malpighi, paraphrasing Homer

MARCELLO MALPIGHI is generally referred to as being the first histologist The better to understand his several contributions to this subject, especially his discovery of the capillaries, mention should be made of the development of the compound microscope, on which his and the subsequent discoveries of his followers depended

According to Henker,[1] there is reason to believe that the magnifying power of transparent media having convex surfaces was known very early, because a convex lens of rock crystal was found by Layard among the ruins of the palace at Nimrud Seneca also described hollow spheres of glass filled with water as being commonly used for magnifiers The perfect gem cutting of the ancients could not have been attained without the use of magnifiers, and it can be assumed that these artificers made their own magnifiers Convex glass lenses were first generally used to assist ordinary vision as spectacles The spectacle-makers were not only the first to produce glass magnifiers, but they were also the inventors of the telescope and the compound microscope During the Thirty Years' War (1618-1648) the simple microscope was widely known, and Descartes in his "Dioptrique," published in 1637, described microscopes wherein a concave mirror was used in connection with a lens for illuminating the object Antony van Leeuwenhoek (1632-1723), who added to Malpighi's initial description of the capillaries, appears to be the first to succeed in grinding and polishing lenses of such short focus and perfect figure as to render to the simple microscope a better object

The early opticians contended that a compound microscope—that is, one having two lenses so that small objects can be magnified—would never produce images as good as those viewed by means of an instrument of the simple type, but this contention has proved to be erroneous. Although the simple microscope may be improved, it has relatively feeble powers of magnification, and to obtain stronger magnifications the compound form is necessary

Soon after the discovery of the telescope, the compound microscope was invented The inventors were probably the Middelburg lens-grinders, Johann and Zacharias Janssen, the time, about 1590 The microscope had a negative eyepiece It was not greatly improved until 1646, when Fontana described a microscope which had a positive eyepiece This produced much better images and was, most likely, the type of apparatus that Malpighi worked with With it, he was able to describe the capillary circulation, the existence of which Harvey, because of his inferior equipment, was able only to postulate

Marcello Malpighi was born at Crevalcuore, near Bologna He was the son of well-to-do parents In 1645 he entered the University of Bologna as a student of philosophy He was forced to interrupt his studies in 1649 because of the sudden deaths of his father, mother, and his father's mother Being the eldest son of the

[1] Henker Otto Microscope, The Encyclopaedia Britannica, ed 11, vol 18, pp 392-407

family, he was called upon to make settlement of the estate This was rendered difficult by reason of a dispute concerning boundaries which had arisen between his family and the possessors of an adjoining property, the family of Sbaraglia This dispute continued to the end of Malpighi's days and was a constant source of irritation The Sbaraglia family not only brought political pressure to bear to obstruct professorial appointments, but also privately heckled Malpighi and his wife

It was not until 1651 that Malpighi resumed his studies, this time with the idea of studying medicine One of his professors, Bartolommeo Massari, to disseminate the new work of Harvey and the new learning of the English philosophers, occasionally gathered some of the instructors and more mature students at his home This group eventually formed itself into a club, limited its membership to nine, the number of the Muses, and adopted the name, "Corus anatomicus" Stimulated by Harvey's new viewpoint of learning by means of actual observation, members of this club not only met for discussion but soon were dissecting bodies and experimenting on living animals Young Malpighi was soon admitted to the club, and therein learned the foundations for his future work He was making remarkable progress in his studies and in 1653 achieved his doctorate in medicine and philosophy

In 1651 Malpighi married Francesca, the sister of his learned professor, Massari She bore him no children In 1656 Malpighi, who had been busy in medical practice, obtained a chair at the University of Bologna and was made a professor of medicine Meanwhile, in the same year, Ferdinand II Grand Duke of Tuscany, offered him a post in theoretical medicine at the University of Pisa Malpighi accepted the offer and for three years taught at Pisa There, under the guidance of Duke Ferdinand, a brilliant intellectual activity was stimulated and sincere efforts were made by the entire staff at the university to broaden the bonds of natural knowledge

It was at Pisa that Malpighi met Giovanni Borelli and much was to come of their friendship, which lasted many years Borelli also had come to the university in 1656 to fill the position of professor of mathematics at the request of Duke Ferdinand Borelli was twenty years Malpighi's senior and taught him the new mathematics and physics of the school of Galileo Malpighi reciprocated and interested Borelli in anatomic and biologic problems to such an extent that soon Borelli was combining his mathematic talents in the study of the phenomena of living things Borelli's great work, "De motu animalium," published after his death (1680 or 1681) shows the influence of Malpighi Although in later years Malpighi's private life was embittered by the coarse personal attacks of Borelli, they remained close friends for many years Whenever a new idea occurred to Malpighi or whenever he made a new discovery, he always desired Borelli's opinion regarding it

In 1659, because of some difficulties regarding the paternal estates, Malpighi resigned his post at Pisa and returned to Bologna There he was again appointed professor of medicine, and in 1660 he was able to announce privately to Borelli, in two letters, his discovery of the structure of the lung These two letters were printed in 1661 and constituted Malpighi's first published work In these brief epistles, two discoveries of fundamental importance were announced In the first letter he described the vesicular nature of the lung, showing how the divisions of the trachea terminated in the dilated air vesicles He was the first, therefore, to supply an anatomic basis for the true conception of the respiratory process

The second letter contained the first observation of the capillaries and supplied the missing link in the rationale of the circulation of the blood Without the aid of a microscope, Harvey had discovered and proved the existence of the circulation He had further predicted the possibility "that in the limbs and extreme parts of the body the blood passes either immediately by anastomosis from the arteries into the

veins or mediately from the pores of the flesh, or in both ways '' Malpighi, by histologic demonstration, proved the existence of capillary anastomosis between the arteries and veins

In 1665, in a little tract, ''De omento pinguedine, et adiposis ductibus,'' Malpighi came upon another discovery concerning the blood, the demonstration of the red corpuscles This he failed to interpret properly Under the microscope he observed flat red cells in the mesenteric blood vessels of the hedgehog Apparently, he mistook the red blood corpuscles for globules of fat passing from fatty tissues into the current of the blood This observation was later clarified by van Leeuwenhoek who, in 1674 in the ''Philosophical Transactions'' of the Royal Society, gave the first accurate description of the red corpuscles

Besides discovering the capillaries and being the first to observe, if not to describe accurately, the red blood corpuscles, Malpighi worked unceasingly on the structure of the glands and glandular organs He is also regarded as the founder of descriptive embryology, because of his investigations of the chick embryo He also discovered the ''rete mucosum'' or the Malpighian layer of the skin and further proved that the papillae of the tongue are organs of taste (Garrison, p 255)

In his monumental work on the structure of the viscera (''De viscerum structura, Exercitatio anatomica,'' published at Bonn in 1666), Malpighi did much to advance the understanding of the physiology of the liver, spleen, and kidneys In this volume, moreover, is contained the first account of the general enlargement of the lymphatic vessels with nodules in the spleen, more fully described by Thomas Hodgkin in 1832 During his later years, Malpighi spent much of his time in researches on the anatomy of the silkworm and the morphology of plants, in which latter endeavor he is equally famous in association with his researches in physiology In 1684, he had a great misfortune His house in Bologna burned His microscopes were ruined and many of his precious manuscripts were destroyed

In 1691, the new pope, Innocent XII, invited Malpighi to come to Rome as his personal physician He at first refused, but after being urged by the pontiff, Malpighi, being an old friend, accepted He continued to work in that capacity, but soon after his arrival at Rome he became ill In July 1694, he suffered a mild apoplectic attack and on November 28th he suffered a second attack On the following day he passed away

EPISTLE II.—ABOUT THE LUNGS*†

To that very famous and learned Man

ALPHONSUS BORELLIUS

Celebrated Professor of Science at Pisa

By

MARCELLUS MALPIGHIUS

Professor of Medicine at Bologna

THERE is this difficulty and obscurity to be met with in natural things, that there seems to be something in them that is not to be determined altogether by our senses. And so, steadfastly working with very great labour, we may contemplate Nature showing herself in her beginnings, as it were in a volume elaborated through mysteries. And when we try to unravel the obscure things in the viscera of animals, at length by our efforts, and only with great weariness, we conclude that the truth of our observations is made out. We borrow illumination, as if by degrees, from dissection, sometimes of insects, sometimes of perfect animals. For Nature is accustomed to rehearse with certain large, perhaps baser, and all classes of wild (animals), and to place in the imperfect the rudiments of the perfect animals.

And now, most famous man, I will handle the matter more closely. There were two things which, in my epistle about observation on the lungs, I left as doubtful and to be investigated with more exact study.

(1) The first was what may be the network described therein, where certain bladders and sinuses are bound together in a certain way in the lungs.

(2) The other was whether the vessels of the lungs are connected by mutual anastomosis, or gape into the common substance of the lungs and sinuses.

The solution of these problems may prepare the way for greater things and will place the operations of Nature more clearly before the eyes. For the unloosing of these knots I have destroyed almost the whole race of frogs, which does not happen in that savage Batrachomyomachia of Homer. For

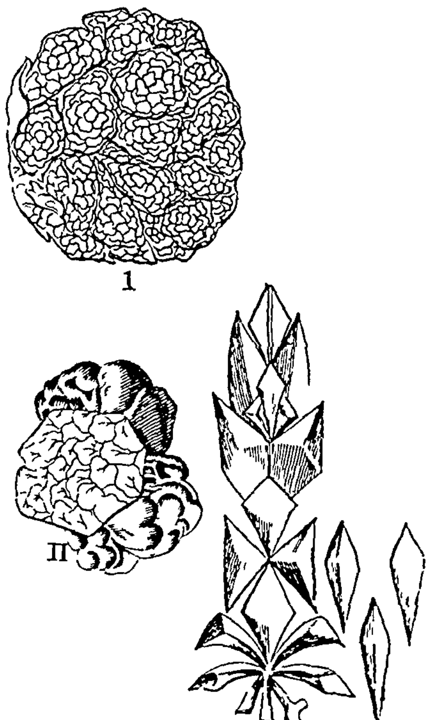
*In his second letter to Borellius, Malpighi describes his discovery of the capillary circulation.—F. A. W., 1940.

†*De pulmonibus observationes anatomicae*. Bologna, 1661. Translated by James Young, M.D., Proc. Roy. Soc. Med. (Part I) 23: 7-11, 1929-1930.

in the anatomy of frogs, which, by favour of my very excellent colleague, D Carolo Fracassato, I had set on foot in order to become more certain about the membranous substance of the lungs, it happened to me to see such things that not undeservedly I can better make use of that (saying) of Homer for the present matter—

"I see with my eyes a work trusty and great"

For in this (frog anatomy), owing to the simplicity of the structure, and the almost complete transparency of the vessels which admits the eye into the interior, things are more clearly shown, so that they will bring the light to other more obscure matters



Tabula I

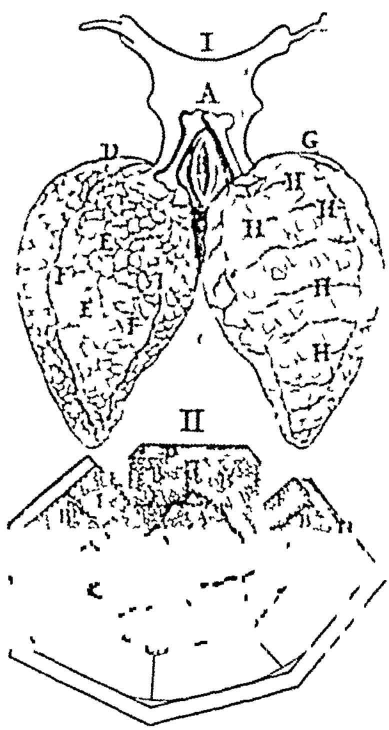
Fig I Outermost piece of dried lung showing the rete

Fig II Interior vesicles and sinuses sketched with portion of the interstitium in the upper part The beginning and complete prolongation could not be exhibited to the eye by the picture

Fig III Adaptation over the trachea and the pulmonary vessels which also, parted from their usual site, are shown for easier understanding

In the frog, therefore, the abdomen being laid open lengthwise, the lungs, adhering on each side to the heart, come forth They are not slack as in other animals, but remain tense for the animal's requirements They are nothing more than a membranous bladder, which at first sight seems to be spattered with very small spots, arranged in order after the fashion of the skin of the dogfish—commonly called Sagrino In form and surface protuberances it resembles the cone of a pine but internally and externally a certain texture of vessels diversely prolonged is connected to-

gether, which, by the pulse, by contrary movement, and the insertion of the vein, are pulmonary arteries. In the concave and interior part of this (bladder) it almost fades into an empty space devoted to the reception of air, but it is not everywhere smooth but is interrupted by the occurrence of alveoli. These are produced by membranous walls raised to a little height. They are not all of this shape, but when the walls are produced out in length and width and connected together, the bays (sinuses) are formed almost into hexagons, and bent at the corners of the sinuses the membrane is extended a little as an infundibulum is constituted, and thus the lungs of the smaller frogs are fashioned. But in those which are larger, the walls are raised higher, and from the middle of the enclosed floor three come out very visibly increasing. The partitions in the smaller frogs are almost unobservable, but those in the bigger ones are bound into three other sinuses as they divide the greater sinus very much. The area, or the floor of the sinuses, admits the vessels spoken of above and the artery itself sometimes ends inconspicuously, fork-like in the middle, but



TABULA II

Fig I Showing lungs of frogs with trachea attached (A) Larynx, which is semi cartilaginous (B) Rima, which is accurately closed and opened at the animal s need Air being enclosed, it keeps the lungs expanded (C) Site of the heart (D) External part of the lung (E) Prolonged rete of the cells (F) Prolongation of the pulmonary artery (G) Concave part of the lung divided through the middle (H) Prolongation of the pulmonary vein running through the apices

Fig II Containing the most simple cell without the intermediate walls (magnified) (A) Interior floor of the cell (B) Parietes separated and bent (C) Trunk of pulmonary artery with attached branches, as if ending in a network (D) Trunk of pulmonary vein wandering with its branches over the slopes of the walls (E) Vessel in the bottom and corners of the walls with the ramifications of the rete continued

further on is spread out at the greater passage and sometimes manifestly produces another branch, but the vein glides down the inner slopes of the walls and is mingled with these, and, the branches having been sent down through the walls, at length runs into the area

Observation by means of the microscope will reveal more wonderful things than those viewed in regard to mere structure and connection for while the heart is still beating, the contrary (i e, in opposite directions in the different vessels) movement of the blood is observed in the vessels,—though with difficulty,—so that the circulation of the blood is clearly exposed This is more clearly recognized in the mesentery and in the other greater veins contained in the abdomen

Thus, by this impulse, the blood is driven in very small (streams) through the arteries like a flood into the several cells, one or other branch clearly passing through or ending there Thus the blood, much divided, puts off its red colour, and, carried round in a winding way, is poured out on all sides till at length it may reach the walls, the angles, and the absorbing branches of the veins The power of the eye could not be extended further in the opened living animal, hence I had believed that this body of the blood breaks into the empty space, and is collected again by a gaping vessel and by the structure of the walls The tortuous and diffused motion of the blood in divers directions, and its union at a determinate place offered a handle to this But the dried lung of the frog made my belief dubious This lung had, by chance, preserved the redness of the blood in (what afterwards proved to be) the smallest vessels, where by means of a more perfect lens, no more there met the eye the points forming the skin called Sagrino, but vessels mingled annularly And, so great is the divarication of these vessels as they go out, here from a vein, there from an artery, that order is no longer preserved, but a network appears made up of the prolongations of both vessels This network occupies not only the whole floor, but extends also to the walls, and is attached to the outgoing vessel, as I could see with greater difficulty but more abundantly in the oblong lung of a tortoise, which is similarly membranous and transparent Here it was clear to sense that the blood flows away through the tortuous vessels, that it is not poured into spaces but always works through tubules, and is dispersed by the multiplex winding of the vessels Nor is it a new practice of Nature to join together the extremities of vessels, since the same holds in the intestines and other parts, nay, what seems more wonderful, she joins the upper and the lower ends of veins to one another by visible anastomosis, as the most learned Fallopius has very well observed

But in order that you may more easily get hold of what I have said, and follow it with your own sight, tie with a thread, just where it joins the heart, the projecting swollen lung of an opened frog while it is bathed on every side with abundant blood This when dried will preserve the

vessels turgid with blood. You will see this very well if you examine it by the microscope of one lens against the horizontal sun. Or you may institute another method of seeing these things. Place the lung on a crystal plate illuminated below through a tube by a lighted candle. To it bring a microscope of two lenses and thus the vessels distributed in a ring-like fashion will be disclosed to you. By the same arrangement of the instruments and light, you will observe the movement of the blood through the vessels in question. You will yourself be able to contrive it by different degrees of light which escape description by the pen. About the movement of the blood, however one thing shows itself, worthy of your speculation. The auricle and the heart being ligatured, and thus deprived of motion and the impulse which might be derived from the heart into the connected vessels, the blood is still moved by the veins toward the heart so that it distends the vessels by its effort and copious flow. This lasts several hours. At the end, however, especially if it is exposed to the solar rays it is agitated, not by the same continued motion, but, as if impelled by changing impulses, it advances and recedes fluctuating along the same way. This takes place when the heart and auricle are removed from the body.

From these things, therefore as to the first problems to be solved from analogy and the simplicity which Nature uses in all her operations it can be inferred that that network which formerly I believed to be nervous in nature, mingled in the bladders and sinuses, is (really) a vessel carrying the body of blood thither or carrying it away. Also that, although in the lungs of perfect animals the vessels seem sometimes to gape and end in the midst of the network of rings, nevertheless, it is likely that, as in the cells of frogs and tortoises, that vessel is prolonged further into very small vessels in the form of a network, and these escape the senses on account of their exquisite smallness.

Also from these things can be solved with the greatest probability the question of the mutual union and anastomosis of the vessels. For if Nature turns the blood about in vessels, and combines the ends of the vessels in a network, it is likely that in other cases an anastomosis joins them, this is clearly recognized in the bladder of frogs swollen with urine, in which the above described motion of the blood is observed through the transparent vessels joined together by anastomosis, and not that those vessels have received that connection and course which the veins or fibres mark out in the leaves of nearly all trees.

To what purpose all these things may be made, beyond those which I dealt with in the last letter concerning the pulmonary mixing of the blood, you yourself seemed to recognize readily, nor is the opinion to be lessened by your very famous device, because by your kindness you have entrusted me with elaborate letters in which you philosophised subtly by observing the strange portents of Nature in vegetables, when we wonder that apples

hang from trunks not their own, and that by grafting of plants the processes have produced bastards in happy association with legitimates We see that one and the same tree has assumed diverse fashions in its branches,—while here the hanging fruits please the taste by a grateful acidity, there they fulfill every desire by their nectar-like sweetness, and you furnish credibility to the truth at which you wondered when in Rome, that the vine and the jasmine had come forth from the bole of the Massilian apple He who cultivated the gardens with a light inserted fork made these clever things with bigger branches, and he taught the unreluctant trees the bringing forth of divers things About this matter Virgil in the Georgics fitly sang —

> "They ingraft the sprout from the alien tree
> *And teach it to grow from the moist inner bark* "

You lay bare the secret of this wonderful result by your philosophising method, for we might consider the acid juice of the Massilian apple sweetens to the nature of pure wine as far as the particles of that juice may run through the small openings of the trunk proper, but not in the same way can they come up into the continued tubules of the vine Here, stirred by their own motion, and torn away beyond their usual order by the impulse of those following after, and broken up, they must conform themselves to the superinduced form of the passage, and put on the new nature by which the vine or jasmine is brought forth Nature pursues a like mode of operation in the lungs, *for the turbid blood returns from the ambit of the body,* widowed elsewhere of particles, to which a new humour from the subclavian vein is added to be perfected by the further action of Nature This happens in order that it may be arranged and prepared into the nature of particles of flesh, bone, nerve, etc , while it enters the myriad vessels of the lungs It is conducted into divers very small threads Thus a new form, situation, and motion is prepared for the particles of the blood, from which flesh, bone, and spirits may be formed The trustworthiness of your saying is increased by the like structure of the seminal vessels as if a certain nutrition of the living animal were also its regeneration

I have put these few little observations into a letter that I might increase the things found out about the lungs If I have set in motion all the point of my observations I have owed the addition to the frog You will bring out the truth and dignity of these matters by your authority and contrivance Meantime, apply yourself happily to philosophy, and may you go on to render me altogether happy by increasing a little my very unimportant thoughts of your writings "De Animalium Motu "

Farewell!

Bologna, 1661

1664

NIELS STENSEN

ON THE MUSCULAR NATURE OF THE HEART

NIELS STENSEN

(Courtesy Annals of Medical History)

NIELS STENSEN

(1638 1686)

NIELS STENSEN or "Nicholas Steno,"[1] as he was also called, was born on January 20, 1638, in Copenhagen, Denmark His father, Steen Pedersen,[2] was a goldsmith who died before Niels was six years of age When his mother remarried, the boy was sent to live with his grandparents and it may be assumed that they provided him with a tutor in order that he might qualify for training at a university

In 1656 Stensen entered the University of Copenhagen For his preceptor, who also was to act in the capacity of adviser, he chose Thomas Bartholin (1616-1680), the esteemed professor of anatomy He not only studied anatomy but also devoted much time to the mathematical sciences At the university he learned Hebrew, which was to be a great asset to him in later years when he became affiliated with the Roman Catholic Church His later published works also show that he was proficient in Greek, Latin, French, German, Dutch, Italian, and English

In 1658 the classes of the University of Copenhagen were interrupted by the Swedish invasion of Denmark The Swedes besieged Copenhagen Stensen, in the short war, held the commission of corporal in the student regiment, with eighty-six scholars under his charge

After spending three years at the University of Copenhagen, Stensen went to Amsterdam, where he studied anatomy under Professor Gerhard Blasius (1626-1682) During his first year at Amsterdam, Stensen discovered the duct that bears his name, the secretory duct of the parotid gland, which he found in the head of a sheep he had dissected Not long afterward, Stensen communicated his discovery to his preceptor, Bartholin, in a letter written from Leyden on April 22, 1661

The letter provoked a quarrel with Blasius who claimed the discovery as his own There is no evidence to support the claim of Blasius, and Jean van Hoorne (1621-1670) of Leyden named the duct after Stensen Because of this quarrel, Stensen left the University of Amsterdam to continue his studies at the University of Leyden, where he worked under Van Hoorne and the celebrated Frenchman, François de la Boe Sylvius (1614-1672) Not long afterward Sylvius was able to demonstrate Stensen's duct in man

Stensen next began to investigate the glands of the eye, and in the latter part of 1661 he published an account of the glands of the eye and the vessels of the nose

From 1662 to 1663 Stensen was busy investigating the muscles In 1664 the death of his stepfather called him home to Copenhagen When he returned to his native city, he published a most important work "De musculis et glandulis observationum specimen, cum epistolis duabus anatomicis" (Hafniae, Lit M Godiecenius, 1664) He dedicated this work to King Friedrich III of Denmark, to whom he had been recommended by Bartholin

The observation, recorded in the aforementioned work, that the heart was composed chiefly of muscle fibers was one of the great anatomic discoveries of his era, and we consider it a special privilege to be able to present Stensen's description of the heart in translation

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[1]Other variations of Stensen Stenon Stènone Stenonis Stenonius
[2]Lutz spelled this name 'Peterson Miller, "Pedersen'

The importance of this discovery, according to Miller, was noted in 1665 by a contemporary, de Hedovvile, who said of it, "This observation overthrew a system to which medicine clung most tenaciously," and Albrecht von Haller (1708-1777), the great physiologist, in 1771, referred to this work as a golden book which contained the rich seed for new discoveries[3]

Lutz referred to Kurt Sprengel (1766-1833) as recording that Giovanni Borelli (1608-1679) claimed in 1680 that he had observed the structure of the heart in 1657, while he was working with Malpighi The only other person to consider the heart a muscle before Stensen was an unknown Alexandrian who wrote a book on the heart which is placed among the Hippocratic works Following Stensen, Richard Lower (1631-1691), in 1669, also demonstrated the muscular nature of the heart

In 1665, Stensen went to Paris, where he continued his anatomical work and also performed dissections at the École de Médecine During this year, Stensen gave a lecture on the anatomy of the brain According to some authorities, modern neurologic observations received their impetus from Stensen's observations which are found in his lecture on the brain

Stensen traveled to Southern France in the latter part of 1665 and spent the following spring in Italy In Italy, he was kindly received and Grand Duke Ferdinand III, who, as we have noticed in our account of Malpighi, had done many things for the cause of science, appointed Stensen one of his physicians and gave him a pension and a residence Stensen then followed the court which moved to the surrounding Italian cities In 1667, at Florence, he published an extensive work on the muscles, "Elementorum Myologiae specimen seu musculorum descriptio geometrica"

On November 2, 1667, Stensen, who had given the matter considerable thought, was converted from Lutheranism to Roman Catholicism In December of that year the Danish King, Friedrich III, offered him a professorship at the University of Copenhagen Stensen wrote to the King asking him if he was willing to accept his new religious preference He received no reply and on February 2, 1670, the King, who had been very ill, died In 1672 the new King, Christian V, commanded Stensen to return to Denmark to assume his professorship, which he did His position as a Roman Catholic professor in a Lutheran university was precarious For some reason, probably his growing attachment to the Roman Catholic Church, he resigned his professorship in 1674 He then returned to Italy and devoted himself exclusively to the work of the church

In addition to his anatomic discoveries, Stensen made many interesting geologic observations He compared fossil teeth found in the Tuscany deposits with the teeth of the living shark He compared deposits of rocks wherein fossils could be found with deposits wherein no fossils could be found, and came to the conclusion that the earth was at one time completely submerged in water Stensen also laid the foundation for structural geology, believing from his studies that the movements of the earth were the cause of origin of vertical strata of rock which once had been horizontal

When Stensen returned to Italy he assumed charge of the education of the son of Grand Duke Cosimo III

In 1675, Stensen was ordained a priest, and, in 1677, he was consecrated Bishop of Titiopolis Jean Frederick, Prince of Brunswick, called him to his court shortly thereafter. Pope Innocent XI allowed him to leave Italy appointing him "Apostolic Vicar for the Northern Missions" Stensen spent the remainder of his life partly at the court of Jean Frederick and partly in visiting Roman Catholic missions in Germany He died at Schwerin on November 25, 1686, and is buried in the Basilica of Saint Lawrence in Florence, Italy

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[3]See page 167, biographic sketch of Albrecht von Haller

NICOLAI STENONIS

DE

MUSCULIS

ET

GLANDULIS

Observationum Specimen.

Cum

Epistolis duabus Anatomicis

AMSTELODAMI,

Apud PETRUM le GRAND, [illegible]

ON THE MUSCULAR NATURE OF THE HEART †

IF ONE desires to apply the word muscle to the heart, one must first demonstrate the proposition that *the heart is truly a muscle* This is deduced from more detailed facts which I shall shortly present

In all the substance of the heart there occurs nothing except *arteries, veins, nerves, fibers and membranes* And in the muscle, except the above mentioned, there occur no others I omit fat and bone, because all hearts do not have this, and no muscle has it Whoever of the anatomists should demonstrate fibers distinctly in the parenchyma should attribute it not to the senses but to genius I saw in certain splendid preparations linear structures running on the surface of the heart, which however were not lymphatics, but dissection showed them to be truly nerves A nervous plexus was demonstrated by Fallopius at autopsy, who believed that they were dispersed not only over the surface but that they penetrated into the interior

Among none of the fibers of the heart does one meet, in examination fibers of which the center is not fleshy, and both extremes tendinous, this moreover is common to all the fibers of the muscle

And thus everything is in agreement, and truly muscle may be attributed to the heart, nor can it be otherwise, *truly the heart merits the name of muscle, because it has tendons and flesh and nerves*

This the great Hippocrates appears to directly oppose nevertheless his words when compared with ours would seem to be in intimate agreement, he said truly that the strength of a muscle was not in the tendon but in the flesh Whatever really was the opinion of this author, his words are only slightly different from ours

If it be true, and the truth of an observation is dependent upon its correctness, that in the heart nothing is lacking which would make it a muscle, and not denying that muscle has been demonstrated in the heart, the heart thus is not a substance *sui generis,* and so cannot generate certain substances as heat (fire), innate warmth, the seat of the soul, nor can it produce certain humors as blood, certain spirits or vitality But however that may be, I am examining solely the substance, thus, from the fibers proceeds all movement of the heart, occurring as a phenomenon of its own , which subject, however, I wish to largely omit at present and defer a consideration of it until a later time

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*Stensen, Niels *De musculis et glandulis observationum specimen cum epistolis duabus anatomicis,* Amstelodanum, OP Le Grand, 1661 90 pp Translated by Maurice N Walsh, M D, Mayo Clinic

†Only such a portion of Stensen's book as deals with the *musculature* of the heart has been translated —Translator

1705

WILLIAM COWPER

DESCRIPTION OF AORTIC INSUFFICIENCY

WILLIAM COWPER

(Courtesy Charles C Thomas)

WILLIAM COWPER

(1666 1709)

WILLIAM COWPER, sometimes written as Cooper, was born in Petersfield, Sussex, England, in 1666 He was the youngest son of Richard Cowper Young Cowper at the age of sixteen was apprenticed to William Bignall, an English surgeon who resided in London At a later date, it is known, he continued his apprenticeship under John Fletcher

In March, 1691, Cowper became free of the Company of Barber Surgeons of London He settled in London, devoted himself to the study of anatomy, and three years later he published a work entitled "Myotomia Reformata, or a New Administration of the Muscles of the Human Bodies, wherein the true uses of the muscles are explained, the errors of former anatomists concerning them confuted and several muscles not hitherto taken notice of described to which are subjoined a graphical description of the bones and other anatomical observations" [London, 1694]

This was a cleverly executed work and no doubt was one of the reasons that led to Cowper's election to a fellowship of the Royal Society in 1696

In 1698 Cowper published a beautiful atlas with an original English text and the following long explanatory title page "The Anatomy of the Human Bodies, with figures drawn after the life by some of the best masters in Europe, and curiously engraven in one hundred and fourteen copper plates, illustrated with large explications, containing many new anatomical discoveries and chirurgical observations To which is added an introduction explaining the animal oeconomy with a copious index by William Cowper Oxford Printed at the Theater, for Sam-Smith and Benj Walford, printers to the Royal Society, at the Princess Arms in St Paul's Church Yard, London, MDCXCVIII "

The publication of this work brought a storm of protests from Godfrey Bidloo, the famous Dutch anatomist Bidloo in no uncertain terms asserted that this work was a plagiarism of his "Anatomia corporis humani, centum et quinque tabulis ad vivum delineatis " Bidloo had originally published this work at Amsterdam in 1685 In 1700, with much bitterness, he called the attention of the medical profession to Cowper's illegal use of his work in a fifty-four-page pamphlet, "Gulielmus Cowper criminus literarii citatus corum tribunali "

In the following year, Cowper replied with his satirical pamphlet, "Eucharistia," in which he stated that the figures for the atlas originally had been drawn for Swammerdam, that the English publishers had purchased the impressions and that he had written entirely new descriptions for the English edition

The truth of the matter was that either Cowper or the publisher had pirated the 105 plates from Bidloo's anatomy Cowper had supplied nine additional plates He had increased the usefulness of Bidloo's plates by adding several references to them The original drawings had been made by the famous Belgian artist, Gerard de Lairesse (1641-1711) A controversy exists concerning the identity of the engraver of the copper plates, but usually he is considered to have been Lairesse Cowper, however, had supplied an original English text, and in an obscure place in a long preface he had said that the engravings "were sometime since Published by Dr Bidloo, now Professor of Anatomy in the University of Leyden "

In spite of the quarrel, Cowper maintained his prestige He was the outstanding British anatomist of his era and among other things became the teacher of the famous surgeon and anatomist, William Cheselden (1688 1752) In 1702 Cowper published his description of the two glands whose ducts, in man, open into the membranous urethra Although these glands are now known as "Cowper's glands," they were originally described by Jean Méry (1645 1722) in 1684

Cowper contributed several interesting papers to the "Philosophical Transactions" of the Royal Society In 1687 he confirmed Malpighi's demonstration (1661) of the capillary function of the pulmonary arteries and veins by demonstrating the capillary circulation in the dog and cat Cowper was the first (1705) to describe aortic insufficiency and he paid particular attention, in his description of it, to the slow pulse that accompanies the condition This most interesting account we are reproducing herein As we shall later note, Vieussens in 1715 also described it and Corrigan in 1832 published his classic account of the condition

Cowper suffered from asthma and dropsy in the last years of his life, so that he finally retired from the strain and strife of London to Bishop Sutton in Hampshire to conserve his health and strength Yet his success was only fleeting, for he died a comparatively young man at forty three, on the eighth of March, 1709 A stone, placed in the wall of the little church at Bishop Sutton by his widow, remains to guard his memory

OF OSSIFICATION OR PETRIFACTIONS IN THE COATS OF ARTERIES, PARTICULARLY IN THE VALVES OF THE GREAT ARTERY*

By

WILLIAM COWPER

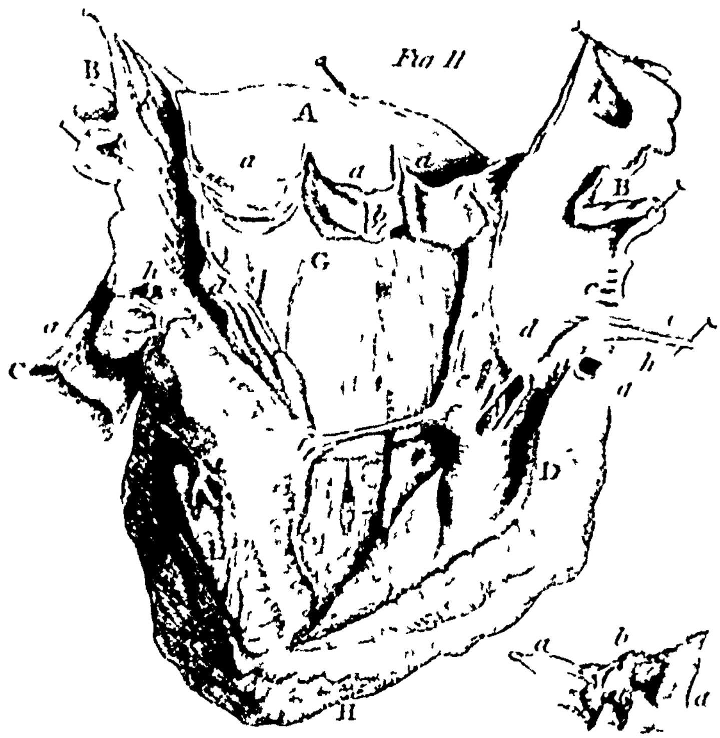
Surgeon, and F R S

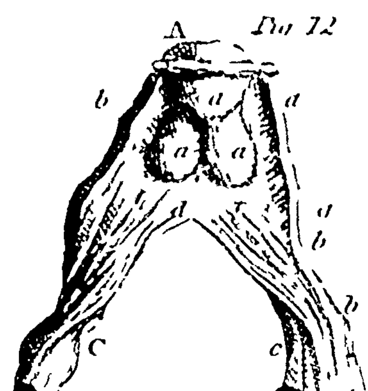
HOW FAR anatomical inquiries inform us respecting the true seats and causes of diseases, which have been ascribed to the want of spirits in some, and of radical moisture in aged people, etc, may be in some measure seen by two observations, among others, published in the Trans No 280 the first there mentioned is of a young gentlewoman, in whom the parietes, or membranes, that compose the trunks of the arteries of the arm near the axilla, being very much thickened, so that the diameter of its bore was lessened to more than a third part of its natural size, insomuch that a part of the trunk of the artery cut transversely, very much resembled a bit of the stem of a tobacco-pipe, its sides were so thick, and its bore consequently so much lessened the other was of the trunks of the arteries of the leg, that were obstructed by petrifactions or ossifications, in a person about the age of 67 Since which, I have met with several of the like instances in aged people, particularly in the legs of an old gentleman, whose toes and foot were sphacelated, in which the ossifications diminishing their channels in some places, and totally obstructing them in others, is made very evident

The dissections of morbid bodies not only instruct us in the seats and causes of diseases, but very often inform us in the true use of parts, as will appear by the following instances The ossification or petrifaction in the great artery, at its rise from the heart, has been so commonly found, that some think it is constant, how it may be in some animals I cannot be certain, but in human bodies I am well assured that whenever it happens, it is a disease, and in some measure incommodes those parts in the due execution of their office, as the following cases will evince but that this paper may be of some use, I shall set down the symptoms before death, which may help our conjectures when the like offers again A thin man about 30, who languished with an ulcer in the thigh, attended with a caries, or rottenness of that bone, at its articulation with the tibia and patella, called the knee where all those bones were affected at length fell into a

*Trans No 299 p 1970 Phil Tr Roy Soc London 5 215-219 1703-1712 (abridged 1809)

true phthisis, and coughed up no small quantity of pus, some months before his death I frequently saw him when he would often offer me his wrist, to feel his unequal pulse, which was wont to amuse him, the artery there missing sometimes one, sometimes two strokes in six or seven, at first he told me he observed it missed but one in ten, but at length those stops became more frequent, especially on any agitation of the body or





mind, though a polypus in any of the great vessels about the heart may induce that symptom, yet its continuance so long before death, shows it owing to some other cause, as appeared on opening the heart and great artery of this person

Fig 11 represents the trunk of the great artery opened and displayed, *aaa,* the three semilunar valves of the aorta, which hinder the blood from

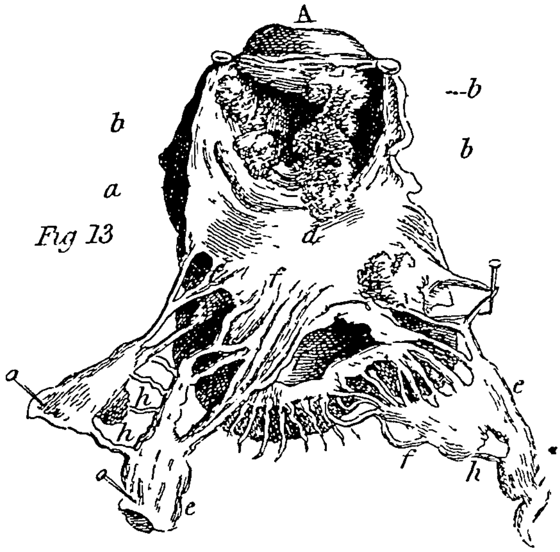


Fig 13

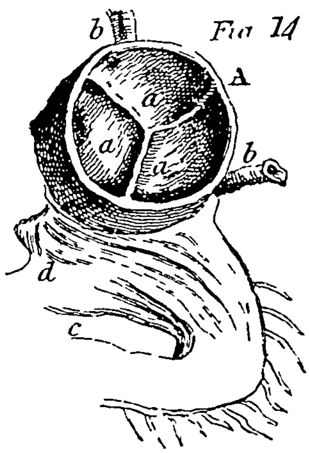


Fig 14

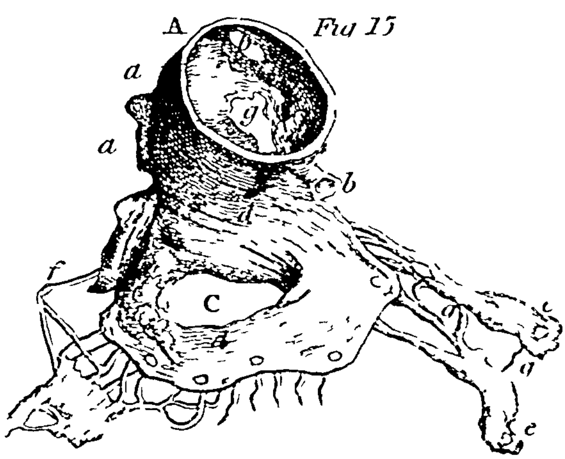


Fig 15

returning to the heart after it is expelled thence by its systole or contraction, these valves in this case were somewhat thicker, and not so pliable as naturally, and did not so adequately apply to each other, as is expressed by *aaa*, Fig 14 Whence it happened sometimes that the blood in the great artery (A, Fig 11) would recoil, and interrupt the heart in its systole But this stubbornness of these valves was owing to a bony or stony substance, marked in the said figure which appeared much plainer when the valves were dry, as represented in the figure beneath, marked with an *, *aa*, the two valves pinned out and dried *b*, the petrifaction or stony body at their junction In this instance I observed the left ventricle of the heart, expressed at GG, DD, *cc*, *ff*, to be a little dilated from its natural size, but was not, by two parts in three so large as the left ventricle of the heart of a subject I have dissected The symptoms, some years before the death of this person, who was about 40 years of age were extraordinary shortness of breath especially on any fatigue with an intermission of one stroke in three of the pulse, the posture of sitting up was more eligible than any other he complained of great faintness and now and then pain about the heart, the extreme parts often cold, which towards his death increased more and more on him, his legs and arms being mortified some hours before On opening the chest, the heart, particularly its left ventricle, was found larger than that of an ordinary ox, and filled with coagulated blood, the valves of the great artery *aaa*, Fig 11, were petrified, insomuch that they could not approach each other, as expressed in Figs 12 and 14, but an orifice, represented at Fig 15, remained always open by the petrifactions *bb*, Fig 13, and *aa*, Fig 15, which had clogged these valves, and hindered their application to each other, as in a natural state is represented in Figs 12 and 14, *aaa*

The explication of the symptoms in both these cases is obvious enough for though the person in the first instance did not die of the same disease as the other, yet the symptoms in his illness plainly showed what must follow from the disorders of these valves, as they are rendered more or less useless, for as their office is to prevent the return of the blood into the heart, in its diastole, by exactly shutting up the passage of the aorta, like the valves in water engines, so if by any accident they are hindered from doing their duty, as they were by the petrifactions mentioned, the consequences must be, not only a regurgitation of blood into the heart, but they baulk its impulsive force, when the muscular fibres in these valves cannot contract, to prepare the passage for the blood of the left ventricle, when it is to be expelled into the aorta Hence the intermissions of the pulse in the first instance may be accounted for In the latter instance, these valves were wholly useless, and the circulation became more difficult, as appeared by the refrigeration of the extreme parts, the mortifications, etc In both these cases, the left ventricle of the heart was dilated proportionably to the bad constitution of these valves, which plainly shows these valves

give such assistance to the heart, as it cannot be without, and that it gradually suffers according to their indisposition

Before these papers were sent to the press, I had an opportunity of observing a like instance of that first mentioned, in an elderly gentleman, about 72, who sometimes had intermissions in his pulse several years before his death, in whom I found divers petrifactions in the mitral and semilunar valves of the left ventricle of the heart

The Explanation of the Figures—Fig 11 represents the left ventricle of the heart opened, etc, AAA, the inside of the aorta slit open to the left ventricle, BB, the bulbous trunk of the vena pulmonalis divided through, and pinned aside, to show *aaa* the three semilunar valves of the aorta, which hinder the blood from returning to the heart, *b*, a small stony substance at the conjunction of two of the semilunar valves, expressed at the ~ below this figure *aa*, parts of the two valves dried, *b*, the petrifaction, as it appears in the dried valves, C, part of the lower trunk of the vena cava, cut off immediately above the liver, *ccc*, the left auricle opened and pinned out, DD, the sides of the left ventricle divided and drawn aside, to show its inside *dd, ee, ff*, GG, *dd*, the mitral valves of the left ventricle of the heart, or arteria pulmonica, divided and turned aside, *ee*, the carneae columnae, whence spring the tendons fastened to the valves, *dd*, expressed by *df* in Fig 13, *ff*, a transverse cord or tendon, by which the columnae carneae are drawn nearer each other in the systole, or contraction of the heart, when the blood is expelled into the aorta, by which the tendons expressed *ff*, Figs 13 and 15, draw the mitral valve laterally, by which means its orifice, *gc* in the said figure, is not only closed to prevent the return of the blood by the vena pulmonalis, but at the same time it opens a passage for the blood of the arteria magna, by withdrawing the mitral valve *d*, Fig 12, from the orifice of the aorta, *aaag*, GG, the internal surface of the left ventricle, where it is somewhat smoother as it leads to the aorta, *gg* the trunk of the coronary vein divided when filled with wax, *hh*, the coronary artery in like manner divided, *i*, one of the trunks of the vena pulmonalis, *kkk*, the three orifices of the trunks of the vena pulmonalis, as they open into the bulbous trunk, expressed at BB, II, the cone of the heart

In Fig 12, A is part of the aorta next the heart, *aaa*, the three semilunar valves, as they appear next the heart in a natural state, when the heart is in diastole, and the blood hindered by these valves from returning to its left ventricle *bb*, part of the basis of the heart cut off, *cc*, the two columnae carneae of the left ventricle, *d*, the mitral valve, *ff*, the tendons springing from the carneae columnae and inserted into the upper and middle parts of the valve as well as to its lower margin, which is better expressed in the following figure *g* the orifice of the aorta completely closed by the application of these three valves to each other

Fig 13 shows the same parts as in the preceding figure, as they appeared when the valves of the aorta were petrified, excepting *a*, which represents a part of one of the valves that was not covered with the petrifaction, *bbb*, the petrifactions on the rest of the valves, † a small petrifaction on the mitral valve, *hhh*, some of the transverse tendons, which draw the carneae columnae to each other, when the heart is in systole, for the more effectual closing the orifice of the mitral valve, expressed here at *q*

Figs 14 and 15 show the same parts represented in the two preceding figures, as they appear viewed towards the heart, when dried and displayed, AA the trunk of the aorta, *aaa* Fig 14, the semilunar valves in a natural state, when the blood in the arteries presses them close to each other, *bbbb*, the trunks of the two coronary arteries cut off, *aa*, Fig 15, the semilunar valves petrified, *c* the orifice of the mitral valve next the vena pulmonalis, *ddd* the internal surface of the mitral valve leading into the left ventricle, *eee*, the columnae carneae, *ff*, their tendons, *gg*, the transverse tendons, which draw the fleshy columns to each other, when the heart is in systole

1708

ANTONY VAN LEEUWENHOEK

HIS CONCEPTION OF "THAT MOTION WHICH WE CALL THE PULSE"

ANTONIUS A LEEUWENHOEK
Regia Societatis Londinensis membrum

ANTONY VAN LEEUWENHOEK

(Courtesy Charles C Thomas)

ANTONY VAN LEEUWENHOEK

(1632-1723)

"The Delphic Oracle"

—Molineux on Leeuwenhoek

ANTONY VAN LEEUWENHOEK did not have the advantages of a university training, but early in life devoted himself diligently to the task of perfecting the single lens microscope He succeeded in making this instrument reflect a clearer and more accurate picture than did the compound microscope of his era

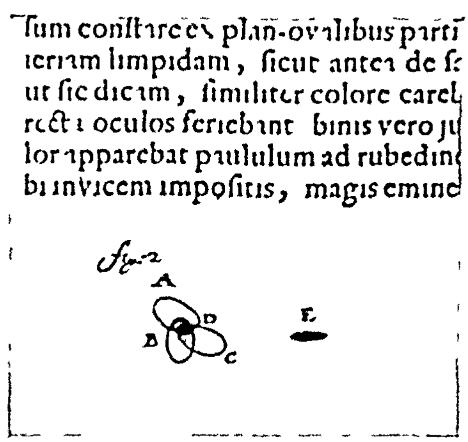
With his newly perfected instrument, he studied most assiduously during the long period of his life, making many noteworthy observations Leeuwenhoek has been accused of making several errors in his discoveries, and it is true that he deceived himself in many of his observations However, considering that he was, in many ways, a pioneer in the vast uncharted field in which he worked, and taking into account the inferiority of his equipment as judged by the excellence of today's microscope, there yet remains a remarkable general truth in many of his observations

Leeuwenhoek was born in Delft, Holland, on October 24, 1632 He came of good Dutch stock Some of his relatives were burghers who manufactured baskets and operated local breweries His father died early and young Antony was sent to school to study for a government appointment He left school at the age of sixteen, however, to become an assistant in a dry goods store in Amsterdam There he remained six years, advancing to the position of cashier and bookkeeper At the age of twenty-two he married a young woman named Barbe de Mey and settled in his native town Delft Five children were born of this marriage, only one of them survived Leeuwenhoek His wife died and he remarried, but no children were born from this second marriage Not much is known concerning Leeuwenhoek for the next twenty years except that he was appointed to the post of chamberlain of the sheriffs of the town of Delft which seemed to be a glorified name for janitor of the City Hall We can assume that much of this time was spent in polishing and in otherwise perfecting lenses for his many microscopes, of which the frames also were made by his own hands

At a later date, Regnier de Graaf, who had immortalized his name by his discovery of the graafian follicle of the ovary, and who was also a native of Delft, became acquainted with Leeuwenhoek and his microscopic discoveries De Graaf, a corresponding member of the Royal Society of London, realizing the importance of Leeuwenhoek's observations, communicated with the members of the society and suggested that they ask Leeuwenhoek to write them concerning his observations Shortly afterward (1673), Leeuwenhoek sent them his first contribution It was written in Dutch, for he knew no other language His first paper was entitled "Some Observations made by a Microscope contrived by Mr Leeuwenhoek in Holland, lately communicated by Dr Regnerus de Graaf" In that paper he wrote

about his microscopic studies of mould of the skin, the flesh, the sting of a bee, the anatomy of the bee, and also the anatomy of the louse During the next fifty years he sent the society about 200 letters regarding his observations, and many of these subsequently were published in the "Philosophical Transactions" We have chosen to reproduce Leeuwenhoek's paper, "On the Circulation of the Blood in Fishes," which he published in the Transactions in 1708 In this paper is found his quaint conception of the pulse, a conception which of course was inaccurate, for he believed that the veins had pulsations and the arteries *none*

In 1674 Leeuwenhoek first observed living protozoa In the following years he described many species which he had demonstrated in well water, in canal water, in infusions of pepper, ginger, and nutmeg, and elsewhere In 1675 he discovered bacteria of various kinds In 1680 he discovered anaerobic bacteria in infusions of pepper, and in a letter describing them he recorded the first microscopic observations ever made on the yeasts in beer That same year he was elected a member of the Royal Society In 1681, this untiring worker discovered the bacteria of the human mouth and intestines, including the spirochetes



The erythrocytes as Leeuwenhoek saw them in 1696,
from his Arcana Naturae

(Courtesy Ciba Symposia)

Leeuwenhoek confirmed Malpighi's discovery of the capillary system He recognized, further, the part the heart played in the circulation Plimmer wrote that Leeuwenhoek said, "I never looked upon the Heart as the maker of the Blood, but only as an Engine that caused the Blood to circulate, driving it forcibly in to the Arteries, and by its opening, giving way for the Blood to come in again out of the Veins"

Malpighi, as we have mentioned, was the first to demonstrate the blood corpuscles, but he mistook them for fat cells Leeuwenhoek was the first to give corpuscles an accurate description He also was the first to mention the leucocytes

Leeuwenhoek also did some remarkable work on spermatozoa, his studies causing him to become a strong opponent of the theory of spontaneous generation

Among his many investigations he produced the first true account of the structure of the optic nerve He also was the first to describe the fibrillated structure of muscle

Interestingly enough, he was the first to use the microscope in a medicolegal inquiry Some material was sent to him which was said to be hair voided from the bladder of a woman He found that it was wool from a stocking

In 1697 Leeuwenhoek was elected a corresponding member of the Académie des Sciences of France, and he made twenty-six communications to that society

Leeuwenhoek was visited in Delft by many royal personages including, in all probability, Charles II of England, who founded the Royal Society, Frederick I of Prussia, Queen Mary of Orange, to whom he gave two of his microscopes, and Peter the Great of Russia

Leeuwenhoek lived to the fine old age of ninety-one He died on August 26, 1723, and was buried in the Oude Kerk in Delft At his decease he bequeathed a collection of twenty-six of his microscopes to the Royal Society

ON THE CIRCULATION OF THE BLOOD IN FISHES, ETC

By

MR LEUWENHOECK,† F R S

I VIEWED the hearts of several fishes, particularly that of the large silver eel, the motion of which lasted near 4 hours, after it was taken out of the body of the fish which motion was very regular for when the blood is protruded out of the heart it is not carried into the great arteries with the same velocity which in that case would be overcharged with the great quantity of blood but the blood thus coming from the heart, is forced into a small white vessel, almost of the shape of a pear and which one would take for a kind of bladder one orifice of which was united to the great artery, and the other to the heart in the latter orifice is a valve, the use of which is to prevent the blood, protruding from the heart into the said vessel, from running back again into it which vessel having been cut across, I observed the inside of it to be furnished with a great many small particles, so that it was in a manner filled with them, and the design of these internal particles I conceive to be that when the blood is protruded into the vessel, by dilating and contracting itself, it may presently force the same into the great artery So that the blood is almost always running with an easy and constant course though at every protrusion it must be in some manner quickened yet that is so insensibly, that it cannot be observed or felt And the case is probably the same in beasts and other large animals

I have formerly communicated some discoveries relating to the circulation of the blood in eels, viz, that the blood, coming out of a great many small vessels in the tail of an eel, is united in one greater blood-vessel, where the fish-bones begin, and where the blood runs through a valve, for I observed that the blood-vein was not only moved in that part where the valve is, but also the parts about it, of the breadth of 4 or 5 hairs breadth, from whence it appeared, that at every protrusion of blood into the heart through the valve, the blood stood still for an instant of time, and then falling through the valve, it ran with great swiftness, and was thickest just at its protrusion out of the valve, but ran thinner or slenderer like the figure of a pear, and the vein that received this protruded blood,

*Trans No 319 p 250 Phil Tr Roy Soc London 5 161-161, 1703-1712 (abridged 1809)

†Spelling of name same as in the original article Note variations from accepted usage —F A W, 1940

was not entirely filled with it, but seemed for a small space to be as it were empty, and its parts contracted, and further observing it, I saw the blood run slowly and leisurely along the same vessel

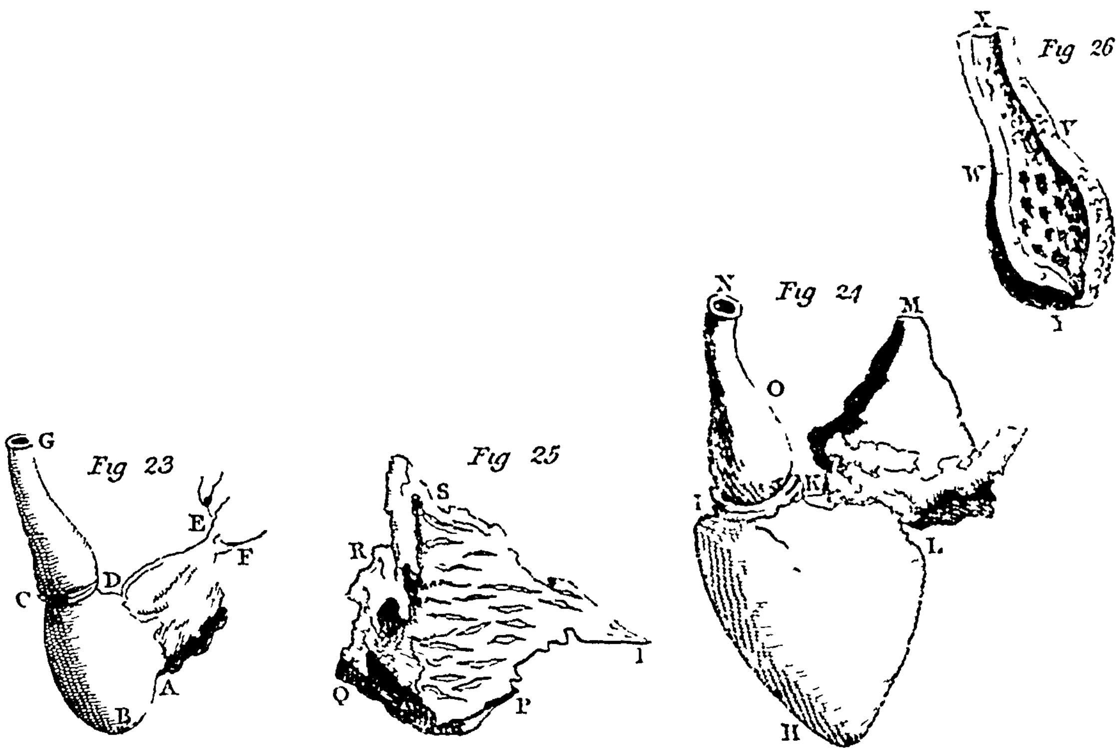
From this observation I imagined, that the same thing happens in the heart of a human creature, viz, that there is a gentle and slow protrusion of the blood out of the heart into that vessel, called the artery, and consequently that there is no such motion there, as is called a pulse, and which is felt in the extreme parts of the body, but that the pulses are only caused by the protrusion of the blood through the valves in the veins, for I never observed any violent or swift protrusion of the blood into the arteries, as often as I have viewed its circulation and though the blood, by the contraction of the heart, be suddenly and hastily protruded out of it, yet it is slowly carried into the artery, whereas, on the contrary, it runs into the heart from the veins with a violent and swift course, from whence it happens, I suppose, that the remaining part of the blood in the veins, being unable to follow with so swift a motion, is, as it were, violently and per saltum drawn or forced through the valves, and that it is this sort of motion which we take for pulses in the arteries

To satisfy myself, in the above observations, I have often viewed that sort of motion in my arm, called the pulse, at the time when my body was without motion and warm, and I judged that the motion, which we perceived in the blood vessel, was not derived from the heart to the hand, but contrariwise from the hand to the arm, and so to the heart from whence I concluded, that, like as in the tail of an eel, there are no valves in the blood vessels, as far as I could perceive, and that a great many small blood vessels, are, as it were, united in that part where the fish bones begin, and make one large blood vessel, where the first valve is, in the same manner in human bodies, a great many single blood vessels running out of the hand, are joined in the arm, where likewise the first valve is, through which the blood at each protrusion falls into the heart, producing what we call the pulse

I have several times observed in the exceedingly small veins or capillary vessels, a little rising or swelling occasioned by a stronger motion of the blood, which I now firmly conclude, to proceed only from the sudden motion or running of the blood through the valves I have also observed, that in sudden frights, and otherwise, one feels such motions at the end of one's fingers, just as if there were valves likewise in them, through which the blood gushes, but these sort of motions, I suppose, do only depend on that quick motion made by the blood, when it runs through the valve in the arm by the hand, to which we give the name of a pulse

In the month of September, having opened an eel, the diameter or thickness of which was about an inch and a half, and having laid open the heart, I could not discover that part which receives the blood out of the great vein in order to bring it into the heart But that I might the better dis-

cover that part, I prepared a little glass tube, and put it into the great vein at a little distance from the heart, and then blew some air into the said vein, as much as might take up the space of about half a pea this air passed through the great vein into a little bladder that lay on the side of the heart, and no sooner was the air got into that bladder, but it first contracted, and then dilated itself, so regularly, and in such a manner that when the heart contracted itself, just as if it were going to protrude its blood, the said little bladder with air in it was dilated and continued in such a motion above 5 full hours together though indeed in the last hour it was so faint, that one could but just perceive it, and as for the heart, its motion was discontinued



I also took a pike-fish, about 2 feet long, and opened it immediately while it was in its full strength of life and observed not only the motion of the heart, and the regular motion of that part which receives the blood, and brings it into the heart, but also the motion of that other part, which receives the protruded blood from the heart, and carries it gently into the arteries

Fig 23, shows the heart of a pike, DEFA represents that part into which the blood is brought from the veins, and CDG, that other part which receives the blood from the heart, to carry into the arteries Now when the heart receives the blood which is conveyed into it, it dilates to its utmost roundness, and then that vessel represented by ADEF at that very instant collapses, and discharging its blood into the vessel, CDG, this becomes distended by the sudden pouring in of the blood, and no sooner is

it so dilated, but it contracts again, that it may force the blood into the arteries In short, when ADEF is contracted, and throws the blood into the heart, this is dilated, and when the heart contracts and discharges the blood, CDG is dilated and these three several motions happen in so short a time, and are performed so regularly, that it is quite surprising and from hence we cannot but conclude, that such a motion as this could not be brought about, unless the vessel ADEF had a valve at AD, where it is joined to the heart, which valve is to prevent the blood, that is thrown into the heart, from returning the same way And so likewise there must necessarily be another valve at CD, to prevent the blood, that is protruded from the heart, from flowing back again

Also Fig 24 represents the heart of a salmon, where KLM shows that instrument or vessel that was represented in Fig 23, by ADEF, as INO shows the same as CDG in the said figure

Also the instrument KLM being cut open, to discover with the naked eye, the sinewy parts and their branches, these appeared as in Fig 25, in which QR is the part that was joined to the heart, and is the same that in Fig 24 is represented by KL, in the said Fig 25, we may observe how the sinewy parts and their branches run from QR to T This instrument, or vessel, is very soft in its parts, and it seemingly is not strong

Fig 26 is that vessel dissected, which in Fig 24 is represented by ION, which vessel is exceedingly thick and strong, and like that represented in Fig 25, provided with strong sinewy parts, that when the parts are extended by the blood poured into them, they may be able both in roundness and length to convey the blood into the arteries these parts, by reason of their great numbers, cannot be delineated in such manner as they ought to be

From the whole I conclude, that the heart protrudes the blood gently into the arteries, and that the blood, which flows from the veins into the heart, causes that sudden revulsion, called the pulse, both because it cannot so immediately pass through the valves, and because the veins in that part are a little narrower, by which means there is a kind of stop or intermission in the circulation of the blood and this I conceive is the cause of that motion which we call the pulse *

*It is scarcely necessary to remark that Mr L's conjectures respecting the cause and nature of the pulse are extremely erroneous and absurd

The above footnote probably was written by either Dr George Shaw or Dr Richard Pearson who with Charles Hutton abridged the *Philosophical Transactions* in the edition (1809) from which we have reprinted Leeuwenhoek's communication —F A W 1940

1733

STEPHEN HALES

EARLY EXPERIMENTS ON BLOOD PRESSURE AND BLOOD VELOCITY

STEPHEN HALES

Portrait by Thomas Hudson

(Courtesy Charles C Thomas)

STEPHEN HALES

(1677-1761)

ON SEPTEMBER 7, 1677, Stephen Hales was born at Beckesbourne, Kent, England He was the sixth son of Thomas and Mary Hales His paternal grandfather, Sir Robert Hales, was created a baronet by Charles II

At the age of nineteen, young Hales entered the University of Cambridge as a pensioner of Corpus Christi College He was graduated from that college with the degree of Bachelor of Arts in 1696 He then accepted a fellowship which he held for several years In 1703 he received the degree of Master of Arts In 1704, still at study under his fellowship, he made the acquaintance of William Stukely (1687-1765), who had come to enroll at Corpus Christi College Stukely and Hales became friends Both were interested in natural history, anatomy, and chemistry They spent considerable time together, sometimes collecting fossils and sometimes in quest of butterflies They also studied comparative anatomy and the dissection of frogs, dogs, and other animals Among other activities they found time to repeat many of Boyle's classic experiments

In 1710, Hales was made perpetual curate at Teddington in Middlesex There he was ordained in the ministry and in 1711 he received the degree of Bachelor of Divinity Hales made Teddington his home for the remainder of his long and useful life He was a very faithful minister and his scientific interests were reflected in his curateship Among other things, he aided his parish in obtaining a pure supply of water, helped construct the church lantern, and replaced the timber tower that held the lantern with a tower made of brick He requested that at his decease, his remains be placed beneath this tower

Some time after he was made curate he was married to Mary Newce, the daughter of Dr Newce, rector of Halisham She died in 1721, leaving him no children

In 1718 he was elected a fellow of the Royal Society of London and in 1719 he reported before the society the results of some experiments he had made on the effects of the warmth of the sun in raising the sap in trees

The Royal Society encouraged him to continue his researches, which Hales did with much enthusiasm His combined efforts were published in 1727 in a volume entitled "Vegetable Staticks, or, an account of some statical Experiments on the Sap in Vegetables being an Essay towards a Natural History of Vegetation, also a Specimen of an Attempt to analyse the Air by a great Variety of chemio statical Experiments, which were read at several Meetings of the Royal Society"

Hales published a second edition of this work in 1731 In the preface to the new edition he promised to contribute a second volume containing more of his studies This appeared in 1733 under the title "Statical Essays containing Haemostaticks, or an Account of some Hydraulick and Hydrostatical Experiments made on the Blood and Blood-Vessels of Animals, also an Account of some Experiments on Stones in the Kidney and Bladder, with an Enquiry into the Nature of these anomalous concretions To which is added an Appendix containing Observations & Experiments relating to several Subjects in the first Volume"

From the second volume of Hales' important work it is our privilege to reprint his classical experiments wherein blood pressure and velocity were first measured

In his first experiments Hales placed a vertical glass tube, the first manometer, in the artery of a horse and measured the distance the blood rose to determine its pressure He performed similar experiments upon the sheep and the dog Much later, Jean Marie Poiseuille (1799 1869) and Karl F W Ludwig (1816-1895) were to measure blood pressure by the means of a mercury manometer This in turn was to be followed by the kymograph of Ludwig and led to the present graphic methods now in use

Hales estimated the blood pressure in man to be about 7½ feet, expressed according to his system of measurement This is somewhat high, of course, but when we consider the crudity of his instruments and the fact that his work was done on experimental animals only, it seems to be a remarkable approximation

Because of his "Statical Essays," Hales achieved an international reputation In 1733 he received the honorary degree of Doctor of Divinity from the University of Oxford In 1739, in recognition of his many achievements, Hales was awarded the Copley medal That same year he published a very instructive work which he dedicated to the Lords of the Admiralty It was entitled "Philosophical experiments containing useful and necessary instructions for such as undertake long Voyages at Sea, showing how Salt-water may be made fresh, wholesome, and how Fresh Water may be preserved sweet, how Biscuit, corn, &c, may be secured from the Weevil, Maggots and other Insects, and Flesh preserved in Hot Climates by salting Animals whole, to which is added an account of Experiments, and Observations on Chalybeate or Steel waters, with some Attempts to convey them to distant places, preserving their virtues to a greater degree than has hitherto been done, likewise a proposal for cleansing away Mud, &c out of Rivers, Harbours, and Reservoirs "

In 1741 Hales, Sutton, a coffee house keeper, and Martin Triewald, a captain of mechanics to the King of Sweden, had independently invented much-needed ventilators for the purpose of removing contaminated air from the lower decks of ships The story of Triewald's invention was read before the Royal Society in 1742 In 1743 Hales' work on ventilators appeared His apparatus was similar to Triewald's It consisted of a large bellows which sucked out the foul air and could be operated either by hand or by windmill Sutton's apparatus operated on a different principle It drew off foul air by the means of the cook-room fire It was of simpler design and replaced, to a large extent, the machines of Triewald and Hales In a few years ventilators were also used for prisons and hospitals Use of them resulted in reducing the death rate in these institutions, a rate which, hitherto, because of most unsanitary conditions, had been terrifically high

Hales was honored with the friendship of Frederick, Prince of Wales He also was on intimate terms with Alexander Pope, who was a neighbor

He died on January 4, 1761, at the age of eighty-four In accordance with his wishes his remains were placed in a vault in the vestry under the new tower he had constructed for St Mary's Church in Teddington A monument to his memory was erected by the mother of George II, the Princess Dowager of Wales It stands in Westminster Abbey in London

STATICAL ESSAYS:

CONTAINING

HÆMASTATICS;

Or, An ACCOUNT of ſome

HYDRAULIC and HYDROSTATICAL

EXPERIMENTS

MADE ON THE

Blood and Blood-Veſſels of ANIMALS.

ALSO

An ACCOUNT of ſome EXPERIMENTS on STONES in the KIDNEYS and BLADDER; with an ENQUIRY into the NATURE of thoſe ANOMALOUS CONCRETIONS

To which is added,

AN APPENDIX,

CONTAINING

OBSERVATIONS and EXPERIMENTS relating to ſeveral SUBJECTS in the Firſt Volume The greateſt Part of which were read at ſeveral Meetings before the ROYAL SOCIETY

With an INDEX to both VOLUMES.

VOL II.

Deſideratur Philoſophia Naturalis vera & activa, cui Medicinæ Scientia inædificetur Fran de Verul Inſtaur Magna

By STEPHEN HALES, D D F R S
RECTOR of FARRINGDON, HAMPSHIRE, and Miniſter of Teddington, Middleſex

The THIRD EDITION, Corrected

LONDON,

Printed for WILSON and NICOL, in the Strand, T DURHAM, near Charing Croſs, G KEITH, in Grace church Street, and ROBINSON and ROBERTS, Nº. 25, in Pater-noſter Row. 1769

TO THE

KING'S

Most Excellent Majesty

SIR

Your Majesty's gracious acceptance of my former Volume of Experiments, has encouraged me, both further to pursue these natural researches and also to lay the result of them at your feet

The study of nature will ever yield us fresh matter of entertainment, and we have great reason to bless God for the faculties and abilities he has given us, and the strong desire he has implanted in our minds, to search into and contemplate his works, in which the farther we go, the more we see the signatures of his wisdom and power, everything pleases and instructs us, because in everything we see a wise design

As the beautiful fabric of this world was chiefly framed for and adapted to the use of man so the greater insight we get into the nature and properties of things, so much the more beneficial will they be to us the more will our real riches thereby increase, the more also will man's original grant of dominion over the creatures be inlarged

Your Majesty's subjects of Great Britain are allowed, by the candid confession of other nations, to excel in experimental philosophy, which has long been found to be most beneficial to mankind

As the advancement of arts and sciences much depends on the protection of princes, whose patronage they are well worthy of, so we have a pleasing prospect of their flourishing under your Majesty's auspicious favour, whose care and concern for the welfare and prosperity of his people, is in every respect most extensive

That your Majesty, after having long continued a blessing to your subjects in a prosperous reign here on earth, may hereafter enjoy a happy immortality in heaven, is the sincere prayer of,

May it please your Majesty,

Your Majesty's
Most humble and
Dutiful subject,

STEPHEN HALES

AN ACCOUNT OF SOME HYDRAULIC and HYDROSTATICAL EXPERIMENTS MADE ON THE BLOOD and BLOOD-VESSELS OF ANIMALS*

EXPERIMENT I

1 IN December I caused a *mare* to be tied down alive on her back; she was 14 hands high, and about 14 years of age, had a fistula on her withers, was neither very lean nor yet lusty: having laid open the left crural artery about 3 inches from her belly, I inserted into it a brass pipe whose bore was ⅙ of an inch in diameter; and to that, by means of another brass pipe which was fitly adapted to it, I fixed a glass tube, of nearly the same diameter, which was 9 feet in length: then untying the ligature on the artery, the blood rose in the tube 8 feet 3 inches perpendicular above the level of the left ventricle of the heart: but it did not attain to its full height at once; it rushed up about half way in an instant, and afterwards gradually at each pulse 12, 8, 6, 4, 2 and sometimes 1 inch: when it was at its full height, it would rise and fall at and after each pulse 2, 3, or 4 inches; and sometimes it would fall 12 or 14 inches, and have there for a time the same vibrations up and down, at and after each pulse, as it had, when it was at its full height; to which it would rise again, after forty or fifty pulses.

2 The pulse of a horse that is well, and not terrified, nor in any pain, is about 36 beats a minute, which is nearly half as fast as the pulse of a man in health: this *mare's* pulse beat about 55 times in a minute, and sometimes 60 or a 100, she being in pain.

3 Then I took away the glass tube, and let the blood from the artery mount up in the open air, when the greatest height of its jet was not above two feet.

4 I measured the blood as it run out of the artery, and after each quart of blood was run out, I refixed the glass tube to the artery, to see how much the force of the blood was abated; this I repeated to the eighth quart, and then its force being much abated, I applied the glass tube after each pint had flowed out: the result of each trial was as is set down in the following table: in which are noted the greatest heights it reached after every evacuation. It was usually about a minute before it rose to these several heights, and did not rise gradually but would stand during

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several pulses much lower, than what it would at length reach to, so that I often thought it had done rising, when on a sudden it would rise for sometime, 4, 8, 12, or 16 inches higher, where it would stay for sometime, and then on a sudden fall 4, 8, 12, or 16 inches

*These five ounces lost in preparing the artery

By this time there is a pint lost in making the several trials which is not allowed for in this table

The several trials	The quantities of blood let out in wine measure		The several heights of the blood after these evacuations	
	Quarts	Pints	Feet	Inches
1	0	*5 ounces	8	3
2	1	0	7	8
3	2		7	2
4	3		6	6½
5	4		6	10½
6	5		6	½
7	6		5	5½
8	7		4	8
9	8		3	3
10	8	1	3	7½
11	9	0	3	10
12	9	1	3	6½
13	10	0		9½
14	10	1	4	3½
15	11	0	3	8
16	11	1	3	10½
17	12	0	3	9
18	12	1	3	7½
19	13	0	3	2
20	13	1	4	
21	14	0	3	9
22	14	1	3	3
23	15	0	3	4½
24	15	1	3	1
25	16	0	2	4

There was about a quart lost in making the several trials, so there flowed out in all 17 quarts, and half a pint after the last trial, when she expired This whole quantity of blood was equal to 996 18 cubick inches

5 We may observe from this table, that the decrease of the force of the blood in the arteries, was not proportioned to the several quantities of blood which were evacuated, for at the eighth trial, when 7 quarts were drawn off, the height of the blood was four feet 8 inches, after which it decreased in the five following trials to three feet odd inches, sometimes a little lower, and then a few inches higher But at the fourteenth trial, after ten quarts and a pint had been drawn off, it rose again up to four feet, 3 plus one-half inches, and it came nearly to the same height again at the twentieth trial, when thirteen quarts and a pint had been drawn off

6 This disproportionate inequality in the several heights was principally owing to her violent straining to get loose, which made the blood in the fourteenth trial rise higher than it had done in several of the preceding ones

7 About the twentieth trial she grew very faint and uneasy, and breathed quick, the violent straining to get loose, did, by the acting of

most of her muscles, especially the abdominal, impel the blood from all parts to the *vena cava,* and consequently there was a greater supply for the heart, which must therefore throw out more at each pulsation, and thereby increase the force of the blood in the arteries

8 For the same reason, too, it would be somewhat increased in height upon deep sighing, because the lungs being then put into greater motion, and more dilated, the blood passed more freely, and in greater quantity, to the left auricle, and thence to the ventricle

9 This plainly shows how sighing increases the force of the blood, and consequently, proportionably cheers and relieves nature, when oppressed by its too slow motion, which is the case of those who are dejected and sad

10 Hence, also we see evidently, that the blood moves fastest and most freely thro' the lungs when they are in a dilated state for which reason animals when they are near expiring, do usually breathe quick, the lungs then labouring to heave fast, that the languid blood may thereby, have a freer course thro' them, to supply the then almost bloodless pulsations of the heart, as was, we see, the case of this mare when her blood was near exhausted

11 When between 14 and 15 quarts of blood had been evacuated, and thereby the force of that which remained in the vessels greatly decreased, then the mare fell into cold clammy sweats, such as frequently attend dying persons, which shows to how low a state the vital force of the blood is at that time reduced Whence we see, that these faint sweats are not occasioned by a greater protrusive force of the blood at that time, but rather by a general relaxation of the pores, as well as of all other parts of the body And it seems hence probable, that the vigour of the blood in the arteries is much abated, when persons who are not in a dying state, have colliquative sweats, as in violent colic pains, fear, &c

12 Upon opening the *mare's* body, I found little or no blood in the *aorta*, about an ounce in the left ventricle, but none in the right, the *vena porta* and *cava* were full, she bled two or three ounces, but very slowly, and not without pressing the jugular vein, which was opened as soon as she expired

13 There might be about two quarts and three-quarters of blood left in the large veins, which, with what was drawn out at the artery, makes five wine gallons, which at 221 cubick inches to the gallon, amounts to 1105 cubick inches, or 42 2 pounds, which, at a low estimation, may be reckoned the quantity of current blood in a horse, there is, doubtless, considerably more, but it is not easy to determine how much

14 As this experiment shews how much the force of the blood in the arteries is abated by different degrees of evacuation, so it may be of use to direct what quantity to let out at a time in bleeding for whatever the real quantity of the circulating blood be it is certain that the estimate of what can with safety be let out at once must be taken from the propor-

tion which that bears to the whole quantity of blood, which will flow out of the vein or artery of the animal till it dies

15 We see also from this experiment, the reasonableness of the practice of bleeding at several distant times, where it is requisite to take away a great quantity of blood, and not to do it all at once, which would too much weaken the force of the blood For since it was found by several instances in this experiment, that when the force of the blood was much depressed by evacuations, it would be considerably raised again by the action of the muscles, out of whose very fine and long capillary vessels it moves but slowly, as also by the motion of all parts of the *mare*, so the case is doubtless the same, when the vigour of the blood is in any degree rebated in the large vessels, by blood-letting, that vigour will in some measure be in a little time restored again not only by the action of the several parts of his body, whereby the blood would have time to flow in from all parts, to supply the most evacuated vessels whereby there would be a just proportionate evacuation of all parts, but also because the vessels themselves would thereby have time to contract themselves in some proportion to the degree of their evacuation

EXPERIMENT II

1 In January, I caused a *gelding* to be tied down fast on his back, in the same manner as the mare was in the foregoing experiment he was 13 hands high, and 10 or 11 years old but very lame, by reason of a canker in his hoof, he was lean, but somewhat lustier than the mare, and much more lively I fixed the same brass pipe and glass tube, as above, to his left crural artery

2 The blood rushed up the tube at once, to near two-thirds of its greatest height, and then more leisurely, as in the mare It would rise and fall commonly, about an inch at each pulsation of the heart but sometimes two or three inches I let out the blood gradually, as in the mare, and after each evacuation, I refixed the glass tube to the artery, to take the several heights of the blood the result of each trial was, as noted in the following table [See opposite page]

3 When I first fixed the tube to the artery, I stopped the horse's nostrils, so as to make him breathe with great difficulty, which made the blood rise five inches higher, but I could not carry this experiment almost to suffocation, as I would have done, because his plunging obliged me to take the tube from the artery He did not bleed half a pint more after this last trial, before he expired

4 We may observe that as this horse was more lively than the mare, so the blood mounted at the first trial 17 inches higher in the tube, than the mare's blood did, yet there flowed three pints of blood less from the horse than from the mare, one reason of this may be, that as she was 4 inches taller, than the horse, so she was probably proportionably bigger

in size every way, and should therefore have more blood, besides, bulk for bulk, the females are observed to have more blood than the males

5 As the quantity of blood decreased, so would the projectile force of the blood in the tube proportionably decrease, so that it would not rise above a quarter of an inch at a pulse, when the horse grew very faint

6 The great ascents or descents of the blood, *viz* 12 or 15 inches at a time, did not seem to be owing immediately to the more vigorous or faint, faster or slower pulsations or systole of the heart, but by its continued equable beating, seemed rather to be occasioned by a more or less quantity of blood flowing in to supply the left ventricle of the heart

The several trials	The quantity of blood let out		The several heights of the blood after those evacuations	
	Quarts	Pints	Feet	Inches
1	0	1	9	8
2	1		9	8
3	2		9	5½
4	3		8	4
5	4		8	2
6	5		7	8½
7	6		7	1
8	7		7	6½
9	8		7	4½
10	9		6	6½
11	10		6	7¾
12	11		5	11
13	12		5*	8½
	12		4†	5½
14	13		4	4
15	14		3	8
16	14	1	4‡	2
	14	1	3§	2
17	15		3	3½
18	15	1	2	10

*The highest point it would stand at for some time
†The lowest points at which points it would continue for some time
‡The highest point
§The lowest point

7 The horse's pulse beat 40 strokes in a minute, before he was disturbed or tied down, but when the glass tube was fixed to the artery, it beat 65 in a minute, and as the horse grew fainter, the pulse was more and more accelerated, so as to beat an 100 times or more in a minute, whence we see, that the pulse is weak and quick, when the heart is supplied with little blood, which is the case in the hectic fevers, etc

8 And the diastole of the heart must necessarily be proportionably small, for if the heart dilated as much, when a small quantity of blood flowed into the ventricle, as when a large quantity entered it must then consequently be filled partly with air each time, which would soon cause the death of the animal

EXPERIMENT III

1 In December I laid a common field gate on the ground with some straw upon it on which a white *mare* was cast on her right side, and in that

the entering wax, and I did the same also at the orifice of the *aorta*, where the valves called *semilunares* were also propelled inward by the above-mentioned brass vent-pipe

15 And this is the proper cavity of the left ventricle, just before its contraction, for at that instant, the blood flowing in from the auricle has opened the mitral valves inward, while at the same time the contracting arteries repel the blood forcibly against the semilunar valves, but at the instant that the ventricle contracts, the mitral valves are closed, being expelled by the blood outwards, while at the same time the semilunar are by the same action opened outwards, to make way for the compressed blood to rush into the *aorta*

16 So that this piece of wax thus formed, may reasonably be taken to be nearly commensurate to the quantity of blood received into this ventricle at each *diastole*, and is thence propelled into the *aorta* at the subsequent *systoles*

17 Having therefore filled a narrow-mouthed vessel brim-full of water, I immersed the wax in it, then taking it out of the water, I filled the vessel brim-full again, from another vessel, whose capacity was divided into cubick inches, which gave the bulk of the wax, and consequently the capacity of the left ventricle, equal to ten cubick inches

18 I got the quantity of the surface of the sides of this ventricle by laying pieces of paper aptly cut to the irregular form of the several parts of the wax, and then laying those papers under another paper, which was equally divided into little squares of one-fourth inch each, by running a pin thro' both papers at every corner of each square, the under papers being thus marked too, it was easy by numbering their several squares and parts of a square, to come pretty nearly to an estimate of the whole inward surface of the ventricle, which I by this means found to be equal to 26 square inches, deducting one square inch for the area of the orifice of the *aorta*, whose diameter I measured from the injected wax

19 The diameter of the *aorta* just before the coronary artery branches from it, was 1 15 inch, whence its area 1 036 square inch

The diameter of the descending *aorta* 0 93, its area 0 677

The diameter of the ascending *aorta* 0 74, its area 0 369

20 The inward area of the sides of the left ventricle being therefore equal to 26 square inches, the sum of the whole pressure of the blood against all the sides of that ventricle, at the instant when it begins first to contract, so as to sustain the pressure of the arterial blood, will be that surface or area multiplied into the perpendicular height of the blood in the glass tube, *viz* 26 × 114 inches, *viz* 2964 cubick inches of blood

21 And since, according to Dr Jurin's estimate, in Motte's Abridgment of the Transactions, part 2d, page 141, a cubick inch of blood weighs 267 7 grains, these multiplied into 2964, the number of cubick inches, and then reduced into pounds, give 113 22 pounds, which is the sum of the pressure

of the blood, which this ventricle sustains, at the instant when it is going to exert a contractive force, sufficient to propel it with considerable velocity into the *aorta*

22 The scruple avoirdupoise contains 18 25 grains, the ounce 438 grains, the pound 7008 grains

23 The area of the greatest section of this ventricle from apex to base being 6 83 square inches, these multiplied into 114 inches, the perpendicular height of the blood, in the tube, give 778 62 cubick inches of blood, equal to 29 7 pounds, the force of the blood which the muscular fibres in that transverse section of the ventricle must resist

24 The velocity with which the blood is thrown out of the ventricle into the orifice of the *aorta*, may be thus computed, *viz* the capacity of this ventricle being equal to ten cubick inches, and the area of the transverse section of the *aorta* being 1 036, by which dividing the ten cubick inches, the quotient 9 65 is the length of the cylinder of blood, which is formed in passing thro' the *aorta's* orifice, at each *systole*, of the ventricle And a horse's ventricle of his heart contracting, or his pulse beating 36 times in a minute, that is, 2160 times in an hour, then a column of blood so many times 9 65 inches, or 20,844 inches long, or 1737 feet will pass in an hour

25 But the *systoles* of the ventricle during which that quantity of blood is propelled, being estimated to be done in one-third of the space of time between each pulse, the velocity of the blood during each *systole*, will be thrice as much, *viz* at the rate of 5211 feet, *i e*, 0 98 of a mile in an hour, or 86 85 feet in a minute

26 Now this velocity is only the velocity of the blood at its first entering into the *aorta*, in the time of the *systole*, in consequence of which the blood in the arteries, being forcibly propelled forward, with an accelerated *impetus*, thereby dilates the canal of the arteries, which begin again to contract at the instant the *systole* ceases, by which curious artifice of nature the blood is carried on in the finer capillaries, with an almost even tenor of velocity, in the same manner as the spouting water of some fire-engines is contrived to flow with a more even velocity, notwithstanding the alternate *systoles* and *diastoles* of the rising and falling *embolus* or force, and this by the means of a large inverted globe, wherein the compressed air alternately dilating or contracting, in conformity to the workings to and fro of the *embolus*, and thereby impelling the water more equably than the *embolus* alone would do, pushes it out in a more nearly equal spout

27 And since the blood in the finest capillary arteries, presses into the veins with a much more equal velocity, than in the *aorta* and greater arteries, since also the *systole* is supposed to be nearly one-third of the time between pulse and pulse, the other two-thirds of that time must be spent in the contraction of the arteries it may therefore reasonably be concluded that the sum of the dilatation of all the arteries in each *systole*

is equal to about the quantity of two-thirds of the blood, which is thrown out in each *systole*, which in the case of this mare is equal to two-thirds of 10 cubick inches, *viz.* 6.66.

28. This ventricle throwing out 10 cubick inches at a time, will in the 36 pulses of a minute throw out 360 cubick inches, equal to 13.75 pounds, and in an hour 825 pounds weight of blood, nearly equal to the weight of the horse.

29. The area of the transverse section of the *aorta* being as above noted 1.036 inch, and the immediate next divisions of it being in the area of the like section of the descending *aorta* 0.677 inch, and that of the ascending *aorta* being 0.369, we find the sum of the two areas of these ascending and descending branches is greater than that of the trunk they arise from, and accordingly the velocity of the blood will be proportionably abated in them, as also on account of what passes thro' the coronary arteries, before the blood arrives at those two branches, of which the descending *aorta* is considerably the largest, thereby to furnish a greater quantity of blood, in the proportion that all the parts of the body below the heart exceed the bulk of those above the heart.

EXPERIMENT IV.

1. I injected also with wax the left auricle and ventricle of an ox's *heart*, which ox was by guess supposed to weigh about 1600 pounds when alive.

The capacity of this ventricle was equal to 12.5 cubick inches.

The area of the transverse section of the *aorta* equal to 1.539 inch.

That of the descending *aorta* equal to 0.912, that of the ascending equal to 0.85.

2. The pulse of a very gentle cow, which was not terrified nor disturbed, while its pulse was counted, was at the rate of 38 in a minute, nearly the same as that of a horse.

3. The capacity of this ventricle 12.5 being divided by the area of the orifice of the *aorta* 1.539, the quotient 8.1 inches is the length of the cylinder of blood, which is formed in passing thro' the *aorta*, in each *systole* of the ventricle.

4. And an ox's pulse beating, or this ventricle contracting 38 times in a minute, that is, 2280 times in an hour, then a column so many times 8.1 inches, or 18,468 inches long, or 1539 feet, will pass in an hour.

5. But each *systole* of the ventricle being performed in one-third of that time, the velocity of the blood in each *systole* will be thrice as great, *viz.* 4617 feet, *i. e.*, 0.874 of a mile in an hour or 76.95 feet in a minute.

6. This ventricle throwing out 12.5 cubick inches at a time, will in thirty-eight *systoles*, which it performs in a minute, throw out 18.14 pounds, and in an hour, and twenty-eight minutes, it will have thrown out 1600 pounds of blood, a quantity equal to the weight of the ox. But this ox being fat,

a quantity of blood equal to his weight must be longer in passing through its heart, than in the lean horse, Exper. III. No. 27, for the fat of animals has little or no blood in it; whence, lean animals have *caeteris paribus* much more blood in them than fat ones.

EXPERIMENT V.

1. I took an estimate also of the force of the blood in a fat gelt sheep or wether, by fixing glass tubes to the jugular vein and carotid artery, in the same manner as I had done to the horse in Exper. III. The sheep was three years old and weighed ninety-one pounds alive.

2. Its pulse beat 65 times in a minute.

3. The blood rose in the tube fixed to the jugular vein 5 + ½ inches, and 9 inches when the sheep struggled and strained.

4. In the tube fixed to the carotid artery it rose 6 feet 5 + ½ inches.

5. The capacity of the left ventricle of its heart, was equal to 1.85 cubick inch.

6. Its inward surface = 12.35 square inches.

7. Its greatest transverse section = 2.54.

8. The area of the transverse section of the *aorta* = 0.172 square inch, that of the descending *aorta* = 0.094, that of the left carotid artery = 0.012, and of the right = 0.07; they both rose separate immediately from the *aorta*.

9. The inward surface of this left ventricle being equal to 12.35 square inches, this, multiplied by 6 feet 5 + ½ inches, produces 957.12 cubick inches of blood = 36.56 pounds, the weight of blood which this ventricle sustains, just before its *systole* begins.

10. And the area of its greatest transverse section being = 2.54 square inches, this multiplied into 6 feet 5 + ½ inches, the height of the blood in the tube, the product is 196.85 cubick inches of blood = 7.51 pounds, the weight of blood which the fibres in this transverse section of the ventricle must sustain.

11. The capacity of the left ventricle being = 1.85 cubick inch, which divided by 0.172, the area of the transverse section of the *aorta*, the quotient 10.75 is the length of the cylinder of blood, which is formed in passing thro' the *aorta* in each *systole* of the ventricle.

12. And this sheep's pulse beating, or his left ventricle contracting 65 times in a minute, that is 3900 times in an hour, therefore a column of blood so many times 10.75 inches, or 41,925 inches long or 3493.75 feet will pass in an hour.

13. But the *systoles* of the heart, during which that quantity of blood is propelled, being estimated to be done in one-third of the space of time between each pulse, the velocity of the blood during each *systole* will be thrice as much, *viz.* at the rate of 10481.25 feet, *i.e.* 1.98 mile in an hour, or 174.6 feet in a minute.

14 And the ventricle throwing out 1 85 cubick inch of blood each time, that will be 4 593 pounds in a minute, or 91 pounds, a quantity equal to the weight of the sheep in twenty minutes

EXPERIMENT VI

1 Having fixed a tube to the left crural artery of a *fallow doe,* the blood rose 4 feet 2 inches in the tube

2 I injected with wax both auricles and ventricles of the heart, of another doe, and found the capacity of the ventricle equal to 9 cubick inches, and the right auricle and ventricle near as big

3 Timorous animals are observed to have larger hearts than courageous ones, as deer, asses, hares, etc which holds true in the instance of this *doe's* heart *Qu* May not one reason of this be, that the fibres of the timorous are generally more lax than those of courageous animals? On which account the blood passing with less resistance through the lax fibred capillary vessels, it was requisite that the heart should at each pulse throw out a greater quantity of blood, in order to supply its more easy and plentiful flow through the lax capillary arteries into the veins And may not this be the reason why the pulses of young animals, as of children, are found to beat faster than those of grown persons? *viz* because the tender fibres of the coats of their blood-vessels being very lax, they give the less resistance to the flowing blood, whose globules are observed by Leewenhoeck to be all of a size both in great and small animals, whence it was needful to make provision for a proportionably greater supply of it from the heart, by increasing the velocity of the dilatations and contractions of that curious engine, in the formation of which are seen such evident marks of the consummate wisdom of the great Author of nature

4 The area of the transverse section of the *aorta* of this *doe* = 0 476, of the descending *aorta* = 0 383, of the ascending = 0 246, and that of the pulmonary artery = 0 502 But it being not easy to obtain, in that timorous creature, the just number of pulses in a minute, I could not calculate the velocity of the blood, nor the quantity that passes in any determinate time

EXPERIMENT VII

1 I fixed tubes also in the same manner to the jugular vein and carotid artery, of several dogs, for whatever experiment I principally intended to make on any dog, I usually began with fixing a tube first to the jugular vein, and then to the carotid artery, which was the method I used to wash the blood out of the capillary vessels, thereby the better to prepare them for my intended experiments

2 The force of the blood in the veins and arteries is very different, not only in animals of different species, but also in animals of the same kind,

and that not only in those of different sizes and weights, but also in dogs of the same size and weight, and even in the same animal, the force of the blood in its vessels is continually varying, according to the different kinds and quantities of food, the various distances of time after taking food, the more or less plethoric state of the blood-vessels, also from exercise, rest, different states of vigour or vivacity of the animal, and many other circumstances, which may conduce to vary the force of the blood, for the healthy state of animals is not confined to the scanty limits of one determinate degree of vital vigour in the blood but the all-wise Framer of these admirable machines has so ordered it, as that their healthy state shall not be disturbed by every little variation of this force, but has made it consistent with a very considerable latitude in the variation of it Now since this force of the blood is so variable, it is the more requisite to be furnished with a good quantity of observations, thereby to find out, the more nearly, a medium of those forces, not only in the same animal, but also in those of different ages, sizes, and kinds, whence haply some curious observations may arise

3 These great inequalities of the force of the blood, not only in different animals, but also in animals of the same kind, may be seen in the following table in Exper VIII No 12 in which I have set down the weights of most of them, and also in different columns, the height to which the blood rose in tubes fixed to the veins and arteries

4 I observed here, as in the above-mentioned horses, and that when the blood had subsided a little in the tubes which were fixed to the arteries of these dogs, it would as in the horses, rise on a sudden considerably on deep sighing, as also on pressing the dogs bellies hard with the hand, the blood would immediately rise about six inches and subside as much on taking off the hand, and it was the same on several repetitions

5 It may be objected to this method of estimating the force of the blood, that by thus fixing tubes to these large veins and arteries, the course of a considerable stream of blood was for that time stopped, and that consequently the force of the blood must be proportionably increased in all the veins or arteries, and therefore also in the veins or arteries to which the tube is fixed and doubtless in some degree it is so In the sheep the left carotide is nearly 1/4 part of the right carotide and descending *aorta* taken together, and in the dog, Numb 3, it is about 1/10 of them

6 To obviate therefore this inconvenience I fixed tubes laterally to the jugular veins and arteries of the dog Numb 13 in the following manner *viz* I took two cylindrical sticks which were 1/2 inch diameter, and 1 1/2 inch in length and having bored holes through them from end to end something larger than those veins and arteries I then slit them in halves

length-ways, and bored another hole through the middle of one of them into its cavity, into which lateral hole the brass pipe entered, which was, at its other end, adapted to fit another pipe which was cemented to a glass tube. Then having laid the vein or artery bare, I drew a linen cloth under it, to wipe it very dry, and then placed under it one of the above-mentioned slit pieces of wood, laying the vein or artery in its cavity, which was covered with pitch, that was at that instant afresh melted with a small warm iron rod, then pouring melted pitch not very hot, over the vein or artery, I immediately put on the other half of the split wood, which had the hole bored thro' it, and tied them fast together: then entering the very slender point of a pen-knife into the above-mentioned hole, I cut an orifice in the vein or artery, and then immediately fixed the brass pipe and tube to receive the following blood, which rose from the jugular vein of the thirteenth dog, first, six inches, and on straining 9 + ½ inches, and from the artery four feet eleven inches, and would doubtless have mounted higher, if the blood had not made an outlet between the artery and the pitch, so as to prevent its rise, which inconvenience might easily be prevented by proper care, which, if done, would give us the real force of the blood against the sides of the arteries, as it did in this jugular vein.

7. I believe this would be a good method to take the force of the blood in lesser animals, where by reason of the smallness of those vessels it might be difficult to insert pipes into them, which if done, those pipes would have too small a bore for the blood freely to pass through them.

8. I have noted in the following table, Exper. VIII. Numb. 12, the several heights to which the blood rose in tubes fixed to the veins and arteries of animals, as they lay horizontally on their backs, or on one side, in the case of the mare, Exper. III. But when an animal stands on its legs, a column equal to the perpendicular height of the animal, must be added to the several heights of the blood in the glass tubes, in order to estimate the force with which the blood presses against the coats of the blood vessels, at the lower parts of the body, and so in proportion for any other part that is higher. So that these columns of blood in the arteries and veins, communicating with each other, are, on account of their equal heights, equipollent to each other: the progressive motion of them being determined by the energy of the heart. And though valves in tubes in which a fluid is propelled upwards with an equal force, would rather retard than promote its progress, yet in tubes where the fluid does not ascend equably but by reason of frequent motions of the whole machine, it is subject to many agitations, in this case valves are of great importance to check the repercussion and regurgitation of the fluid, and accordingly, the all-wise Framer of animal bodies has provided valves in the veins, to prevent this inconvenience, and that principally in the lower parts of the body, where they are most needed, especially in great motions, and in exerting the muscular force of the body.

EXPERIMENT VIII

1 The blood having risen six feet eight inches from the crural artery of the dog, Numb 1 and to the same height from the left carotide artery of Numb 7 in the table, Numb 12 of this Experiment VIII, I chose to calculate the velocity, &c of the blood of this dog

2 The capacity of the left ventricle of the heart, being injected with wax, was found equal to 1 172 cubick inch

3 Its inward surface equal to eleven square inches, which multiplied into the perpendicular height of the blood in the glass tube, which was fixed to the artery, *viz* six feet eight inches, or eighty inches, gives eight hundred and eighty cubick inches of blood, which press on all sides of that ventricle, when it has contracted just so far, as to sustain and be equal to the force of the blood in the *aorta*

4 These eight hundred and eighty cubick inches multiplied by 267 7, the number of grains in a cubick inch of blood, gives 235,567 = 33 61 pounds

5 The area of the transverse section of the *aorta*, just before the coronary arteries branch off from it, being 0 196 square inch, by which dividing 1 172 cubick inch, the capacity of the ventricle, the quotient 5 978 inches, is the length of the cylinder of blood, which is formed in passing thro' the orifice of the *aorta*, at each *systole* of the ventricle

6 And a dog's pulse being found to beat, or his left ventricle to contract, ninety-seven times in a minute, then a column of blood so many times 5 97 inches long, will be 34,745 4 inches, or 2,895 45 feet long, but the *systoles* of the heart during which that quantity is propelled, being estimated to be done in one third of the time between pulse and pulse, the velocity of the blood during each *systole* will be thrice as much, *viz* 8,686 35 feet, that is at the rate of 1 64 mile in an hour, or 144 77 feet in a minute

7 And the ventricle throwing out 1 172 cubick inch of blood in each *systole*, that is, 4 34 pounds in ninety-seven pulses, the number of pulses in one minute, hence fifty-two pounds a quantity equal to the dog's weight, will pass thro' the heart in 11 9 minutes

8 If according to Dr Keill's estimate, the left ventricle of a man's heart throw out in each *systole* an ounce or 1 638 cubick inch of blood, and the area of the orifice of the *aorta* be = 0 4187, then dividing the former by this the quotient 3 9 is the length of the cylinder of blood, which is formed in passing thro the *aorta* in each *systole* of the ventricle, and in the seventy-five pulses of a minute a cylinder of 292 5 inches length will pass this is at the rate of 1462 feet in an hour But the *systole* of the heart being performed in one-third of this time the velocity of the blood in that instant will be thrice as much *viz* at the rate of 43861 feet in an hour or 735 feet in a minute

9 And if the ventricle throws out one ounce in a pulse, then in seventy-five pulses of a minute the quantity of blood will be equal to 4 4 pounds, 11 ounces, and in 34 minutes a quantity equal to a middle-sized man, *viz* a hundred and fifty-eight pounds, will pass thro' the heart

10 But if with Dr Harvey and Dr Lower we suppose two ounces of blood, that is, 3 276 cubick inches to be thrown out at each *systole* of the ventricle, then the velocity of the blood in entering the orifice of the *aorta,* will be double the former, *viz* at the rate of 146 feet in a minute, and a quantity of blood equal to the weight of a man's body will pass in half the time, *viz* 17 minutes

11 If we suppose, what is probable, that the blood would rise 7 plus ½ feet high in a tube fixed to the carotide artery of a man, and that the inward area of the left ventricle of his heart, is equal to fifteen square inches, these multiplied into 7 + ½ feet gives 1350 cubick inches of blood, which presses on that ventricle, when first it begins to contract, a weight equal to 51 5 pounds

12 That we may the more readily compare the above-mentioned several estimates together, I shall here range them in order in a table

The several Animals		Weight of each		Height of the Blood in the Tube from Jugul		Height of the Blood in Tubes fixed to Arteries		Capacity of the left Ventricle of the Heart	Area of the Orifice of the Aorta	Velocity of the Blood in the Aorta
		Pd	Cu	Inches		Feet	Inch	Cubick Inches	Square Inches	Feet Inch in a Minute
Man			160			7	6	1 659	0 4187	56 55
								3 318		113 3
Horse	1st					8	3			
	2d					9	8			
	3d		825	12 On straining	52	9	6	10	1 036	86 85
Ox			1600					12 5	1 539	76 95
Sheep			91	5½	9	6	5½	1 85	0 172	174 5
Doe						4	2	9	0 476	
Dogs	1st		52	0	6	6	8	1 172	0 196	144 77
	2d		24	5	7	2	8	1	0 185	130 9
	3d		18	5		4	8	0 633	0 118	130
	4	12	8	4		3	3	0 5	0 101	120
	5			4	6	at crural		1 25	0 210	144 28
	6		31			Arter			0 196	
	7		43			6	8	1 172	0 179	156 59
	8					6	6	Tube fixed to the crural artery		
	9			7	14	3	1			
	10		15	5	24	1	6	was very old, and died soon		
	11		37	8½		4	9			
	12		36			6	7			
	13		24	6	9½	4	11	Tube fixed laterally to the left		
	14	37	8			5	8	carotide artery		
	15			5	19	on sucking at the tube				
	16			5½	8	on sucking				
	17		19	5	14	5	2			
	18		35	5		4	7			
	19		32	6	9½	3	11			
	20		23	5	7	4	10			

13 I do not see, by comparing the weights of these animals, and the several quantities of blood which pass thro' their hearts in a given time, that we can thence form any rule that is fixed, for the proportioning the quantities of flowing blood to their different sizes

14 These quantities in larger animals are very disproportionate to the bulk of their bodies, in comparison of what they are in lesser animals as estimated in this table

15 But as in the bigger animals the blood had a longer course to go, and must therefore meet with a greater resistance, so we may observe in this table, by comparing the perpendicular heights of the blood in the tubes fixed to the arteries, that the force of it in the arteries is in the main greatest in the largest animals

The several Animals		Quantities of Blood = to the Weight of the Animal in what time	How much in a Minute	Weight of the Blood sustained by the left Ventricle contracting	Number of Pulses in a Minute	Area of the transverse Section of descending Aorta	Area of the transv Sect of ascending Aorta	
		Minutes	Pounds	Pounds		Square Inches	Square Inches	
Man		34 18	4 38	51 5	75			
		17 6	9 36					
Horse	3d	60	13 75	113 22	36	0 677	0 369	
Ox		88	18 14		38	0 912	0 85	
							Right	Left
Sheep		20	4 593	36 56	65	0 094	0 07	0 012
						0 385	0 246	
							Right	Left
Dog	1	11 9	4 34	33 61	97	0 106	0 041	0 034
	2	6 48	3 7			0 102	0 031	0 009
	3	7 8	2 3	19 8		0 07	0 022	0 009
	4	6 7	1 85	11 1		0 061	0 015	0 007
						0 119	0 7	0 031
						0 125	0 062	0 031
	7	9 9	4 34			0 109	0 053	0 032

16 And supposing the blood-vessels in the man and horse to be equally distributed in all their homologous parts that is, if they are proportionable to their respective weights then the blood must move in them reciprocally as the times, in which quantities of blood equal to their respective weights, pass thro their hearts viz as 60 to 17 minutes

17 So that notwithstanding the arterial blood of a horse is propelled with a greater force than that of a man yet it moves the slower in the horse on account of a greater number of ramifications and a greater length of vessels in the larger animal

18 When I compared the proportion that the area of the transverse sections of the descending *aorta* bears to the flesh and other parts which they supply with circulating blood I found it to be as follows viz having cut the body of a dog asunder at his heart and first weighing each part

sepaiately, and then boilıng them so as to sepaiate the bottes [bones] fiom the flesh, the weight of the bones beıng deducted fiom the flesh, the flesh, etc of the hındei pait was found to weigh eleven pounds, eleven ounces, that of the foiepait seven pounds and two ounces

19 Now the aieas of the tiansveise section of the aiteiies of these five anımals aie by measuiement as follows, *vız*

20

	Aorta	desc	ıscend		
In the Mare	1 036	0 677	0 369		0 412
Ox	1 539	0 912	0 85	by Computation fiom the	0 556
Sheep	0 172	0 094	0 082	ıbove found pioportion of	0 057
Doe	0 476	0 383	0 246	flesh fiom the hındei and	0 233
1st Dog	0 196	0 106	0 075	foie parts	0 064
6th Dog	0 196	0 125	0 093		0 076
7th Dog	0 179	0 109	0 085		0 066

21 In thıs table we find that the aieas of the tiansveise section of the descendıng and ascendıng *aoita's* of the fiist dog aie neaily pioportıonable to the weights of the iespective paits which they supply with blood, and that ın the maie and doe, the diffeience ıs not gieat, but gieatei ın the ox and sheep In estımates of thıs kınd gieat accuiacy ın the pioportıons ıs not to be expected

22 The velocıty wıth whıch the blood ıs thiown out of the left ventiıcle, beıng peifoimed ın one-thiid of the tıme between *sys'ole* and *systole,* the lıke quantıty of blood would move wıth an equable motıon, of one-thiid of that velocıty, thio' the oiifice of the *aoita* ın the space of tıme between each *systole*

23 Sınce ın a man a cylındei of blood of the diametei of the oiifice of the *aoita* and 7 92 ınches long, ıs at each pulse ımpelled thiough a dılatable conıcal aiteiy, ıts velocıty would be gieatly ıncieased by passıng thio' that naiiowei defile, but the aiteiies contınually sendıng off ınnumeiable bianches, the sum of whose oiifices aie consıdeiably laigei than the maın stems, hence the velocıty of the blood must be pioportıonably iebated So that as Di James Keıll, ın hıs *Tentamına Medıco Physıca* p 46, has estımated ıt, the velocıty of the blood at the heait, would be to ıts velocıty ın an evanescent aiteiy as 5233 to 1, ıf ıt had a fiee unembaiiassed couise thiough those capıllaiy aiteiies And sınce the velocıty at ıts passıng fiom the heait to the *aoita* ıs at the iate of 146 feet ın a mınute, takıng one-thiid of that, *vız* 43 2 foi ıts contınued equable velocıty, accoidıng to Di Keıll's estımate ıt would move but 0 00901th pait of a foot, oi 0 1128 ınch ın a mınute, ın the evanescent aiteiies ın that tıme

24 Thıs would be ıts velocıty theie, ıf the blood had as fiee and unembaiiassed a couise thio' the finei capıllaiy aiteiies, as ıt has thio' their largei iamifications But by the followıng expeiıment, ıt ıs found, that the piıncıpal obstiuction to the piogiess of the aiteiial blood ıs ın the capıllaiy aiteiies

1 I slit open with a pair of scissars, from end to end, the guts of a dog, on that side which is opposite to the insertion of the mesenterie arteries and veins, and having fixed a tube 4 + ½ feet high to the descending *aorta* a little below the heart, I poured blood-warm water thro' a funnel into the tube, which descended thence into the *aorta*, with a force equal to that, with which the blood is there impelled by the heart This water passed off thro' the orifices of innumerable small capillary vessels, which were cut asunder thro' the whole length of the slit gut But notwithstanding it was impelled with a force equal to that of the arterial blood in a live dog, yet it did not spout out in little distinct streams, but only seem to ouze out at the very fine orifices of the arteries, in the same manner as the blood does from the capillary arteries of a muscle cut transversely

2 Having provided a pendulum which beat seconds, and pouring in through the tube known quantities of warm water, I found that 342 cubick inches of water passed off in 400 seconds, or 6 6 minutes

3 Then cutting all the mesenteric arteries asunder close to the guts, and taking away the guts, I found that a like quantity of water passed thro' these larger ramifications of the arteries in 140 seconds, or 2 3 minutes, that is, in one-third of the time

4 Then cutting asunder the crural arteries, which were before tied, and cutting off the mesenteric and emulgent arteries, close to the *aorta*, a like quantity of water passed thro' this thus cut *aorta* in 0 308 minutes, that is in 1/21 4, or 0 467th part of the time, in which it passed thro' the capillary arteries of the slit guts

5 There being 342 cubick inches which passed thro' the capillary arteries of the slit guts in 6 6 minutes, that is, thirteen pounds, if it were blood, or 1 969 pound in a minute, and it being estimated in the table [p 147], that 4 34 pounds of blood were thrown out of the heart of the dog Numb 1 in a minute, the above-mentioned 0 969[c] pound is 1/2 2 = 0 454th part of what passes the heart in that time

6 But on weighing all the fleshly and other membranous parts of another dog, through which the arterial blood passes, that is, exclusive of the bones and lungs I found the whole weight to be eighteen pounds eleven ounces, of which the slit gut weighing one pound two ounces, was therefore, 1/16 6 or 0 06th part of the whole and there going 4 34 pounds out of the heart in a minute and 1 969 pounds passing the mesenterick artery in the same time and the slit gut weighing but 18 ounces and all the parts of the body weighing 299 ounces or 18 pounds 11 ounces, therefore, 1 969 pounds pass thro 18 ounces whilst 2 371 pounds pass 281 ounces (for 4 34 − 1 969 = 2 371 and 299 − 18 = 281) but 1 969 18 = 0 1094

and 2374/281 = 0 008474, and 0 1094   00 08474   12 1   91 1* So that bulk for bulk there passed 12 91 times more water through the arteries of these slit guts, than through the rest of the arteries of the body, and that with a force no greater than that of the heart

7 Which may reasonably be attributed to these several causes, as to the much greater fluidity of water than of viscid blood, to the more relaxed state of these arteries in the dead than in the living animal, for tho' the arteries and veins of a dead animal being then freed from the distending force of the blood do contract, yet with equal forces, those of the dead animal will dilate more than those of a live animal but this more plentiful flow of water is principally owing to the great difference there is in size, between these cut capillary arteries, and the succeeding series's of exceeding small ramifications, and that at right angles, thro' which the blood passes in its further progress towards the vein as also to a want of the resistance *of the venal blood, which rising six inches in the tube fixed to* the jugular vein is 1/13 33 or 0 075th part of the force of the arterial blood, and must therefore proportionably retard its motion

8 The diameters of the cut orifices of the arteries thro' which the water passed off, were at a medium, one with another, equal to twice the diameter of a hair, which Dr Jurin, by an accurate estimation, found to be 1/324th part of an inch, hence these arteries, which are 1/162 inch diameter, as they branch off from the mesentericks, spread themselves alternately on each side of the guts, whence meeting again, their inosculating branches form *areola's* like those that are on the leaves of trees and from these thus converging arteries and sap-vessels, there branch off nearly at right angles, without converging any more, much smaller arteries, and from these others again, both at right angles, and like the spread fingers of a hand, in successive series's in their progress towards the veins

9 The diameters of the first series's of these unconverging branches, may in a piece of gut well injected with vermilion, be observed to be of several sizes, from ½ to ⅓ of the arteries whence they rose, and the succeeding ones finer and finer to nearly 1/3240th part of an inch, that is so fine, that only single blood globules can pass them into the veins, here therefore, so viscid a fluid as the blood must needs meet with a very great resistance

10 These reticular converging arteries, by being thus inosculated into each other, both prevent obstructions in them, and also thereby the most plentiful supply the next series of rectangular branches with blood, for if the blood had entered the converging arteries only at one end, its velocity would thereby have been more retarded in going the whole length, than half the length of these converging arteries, by these innumerable convergencies of the arteries, the blood is more blended and mixed, as is plain to be seen in the lungs of frogs

*Several obvious errors appear in this proportion which we reprint exactly as it appeared in the 1769 edition The figure 12 91 is obtained as follows 1 969/18 = 0 1094 and 2 371/281 = 0 008474 and 0 1094   0 008474   12 91   1—F A W 1940

11 From this experiment we see how greatly the velocity of the water is retarded in passing thro' the several branchings of the arteries, notwithstanding the sum of the areas of their transverse section is considerably greater than that of the *aorta* And this retardation must be still greater to the blood which is both a grosser and more viscid fluid than water, and that especially in the extreme capillary arteries which branch off at right angles, and which are about 1/1620th part of an inch in diameter so fine that only single globules of blood can pass them

12 And to this resistance which the blood meets with in passing the capillary arteries, is owing the great difference of the force of the blood in the arteries to that in the veins, *viz* as 10 or 12 to 1

13 For tho' the velocity of the blood at its first entrance into the *aorta,* depends on the proportion the area of its orifice bears to the quantity thrown into it at each *systole,* and also on the number of those *systoles* in a given time yet the real force of the blood in the arteries, depends on the proportion, which the quantity of blood thrown out of the left ventricle, in a given time, bears to the quantity which can pass thro' the capillary arteries into the veins, in that time

14 But the resistance which the blood meets with in those capillary passages may be greatly varied, either by the different degrees of the viscidity or fluidity of the blood, or by the several degrees of constriction or relaxation of those fine vessels, instances of which may be seen in Experiments 15, 16, 17, 18

15 And as the state of the blood or blood-vessels is in these respects continually varying from divers causes, as motion, rest, food evacuations, heat, cold, &c so as probably never to be exactly the same any two minutes, during the whole life of an animal, so nature has wisely provided, that a considerable variation in these, shall not greatly disturb the healthy state of the animal

16 We may make a pretty near estimate of the force of the blood in the capillary vessels in the following manner, *viz* taking the diameter of a blood-globule to be as above 1/3240th part of an inch, which Leewenhoeck has observed to be of the same size both in small and great animals, and allowing these capillary vessels to be a small size larger than the globules, which swim in and are carried along by the *serum* which surrounds them on all sides, we may therefore well suppose one of these vessels to be double the diameter of such a globule *viz* 1/1620th part of an inch, or 0.000617, the periphery therefore of this vessel will be 0.001939 and its area 0.000000298 which multiplied by 80 the number of inches to which the blood rose in the tube when fixed to the artery of the dog Numb 1 gives 0.000239 parts of eighty cubick inches of blood, or of 21.416 grains, equal to 0.515 parts of a grain But the resistance of the blood in the veins of the same dog being found equal to six inches height or 1/13.33d or 0.075 parts of eighty inches this 1/33.33d part = 0.03039 being deducted

out of 0 5118 parts of a grain, the remainder 0 4734 grain is the force with which the blood would be impelled into such a capillary by a column of blood of eighty inches height, supposing it were in a stagnant state, to which also must be added the velocity which the blood has acquired at its first entrance in the capillary vessel, which can be but small, as appears by the great resistance it meets with in the capillary vessels, in this 9th Experiment, Numb 18, whence we see, both from experiment and calculation, that the force of the blood in these fine capillaries can be but very little, and the longer such capillaries are, the slower will the motion of the blood be in them

17 It is observable, that these parallel arteries are not as in the bowels, lungs, and other membranous parts of the body, intermixed with corresponding similar veins, but two different series's of these arteries, arising at right angles from larger arteries, one series from the upper, and the other from the lower parts of the muscles, their parallel arteries do mutually and alternately intermix, whereby the blood is conveyed in them alternately upwards and downwards, and thence flows at right angles into the veins

18 From this very small force of the arterial blood among the muscular fibres we may with good reason conclude, how short this force is of producing so great an effect, as that of muscular motion, which wonderful and hitherto inexplicable mystery of nature, must therefore be owing to some more vigorous and active energy, whose force is regulated by the nerves but whether it be confined in canals within the nerves, or acts along their surfaces like electrical powers, is not easy to determine

19 That a vibrating electrical virtue can be conveyed and freely act with considerable energy along the surface of animal fibres, and therefore on the nerves, is evident from curious experiments, made by that skillful and indefatigable experimenter, Mr Stephen Gray, of which he has given an account in the *Philosophical Transactions,* Numb 417, 422, where he shews that electrical virtues from a glass heated by rubbing, will not only be conveyed along the surface of lines to very great lengths, but will also be freely conveyed from the foot to the extended hand of a human body suspended by ropes in a horizontal posture in the air, and also from that hand to a long fishing-rod held in it, and thence to a string and a ball suspended by it and also that an electrical virtue may be carried along a surface of water

20 And it has been frequently observed, that when some part of the body has upon itching been gently scratched by the nails, there has at the same time been felt in a distant part, a very pungent *stimulus* or sensation answering exactly stroke for stroke to the action of the scratching nails Thus particularly, on scratching a small pimple, a little below the right knee on the outside, a like pungent sensation has been felt on the left

shoulder-blade, and sometimes on that arm some inches below the shoulder and *vice versa* the right shoulder or arm has in like manner been affected, when the scratching has been made near the left knee, but this effect does not always follow there are many instances of the sympathy of the nerves

21 That the animal spirits, whether they act within or on the outsides of the nerves, are elastick, seems probable not only from their great activity and energy, but also from the sudden and strong effects that sulphureous vapours, which are known to destroy elasticity, are found by experience to have on them Thus the fumes of burning brimstone will instantly deprive all animals whatever of life thus also the subtile and most penetrating fumes of fermenting spirituous liquors, are known either to strike those instantly dead who smell to them, or to infatuate or render paralytic for life, those who smell to them in lesser degrees Thus also the sulphureous foetid fumes of burnt feathers, etc have an effect on the *disordered spirits* of those who are in fits Thus also *assa foetida, castor,* &c which abound with a subtile sulphur, are found to be friendly to the spirits of the hysteric, as on the contrary are many fumes most offensive to the spirits of others

22 If the skin be flead off the belly of a live frog, and the abdomen opened on each side, so as that its strait muscles may, by drawing a little on one side, have a strong focal light cast on the inside of them, if in this posture those muscles be viewed thro' a good microscope, the parallel fibres of the muscles are plain to be seen, with the blood running alternately up and down between each fibre, in capillary arteries so fine that only a single globule can pass them If the muscle happens to act while thus viewed, then the scene is instantly changed from parallel fibres, to series's of *rhomboidal pinnulae* which immediately disappear as soon as the muscle ceases to act It is not easy to get a sight of this most agreeable scene, because that on the action of the muscle, the object is apt to get out of the focus of the microscope, but those who are expert in the use of those glasses may readily move them accordingly I have found small frogs best for this purpose, *viz* such as are not above ⅓ or ¼ of their full growth Stimulating the foot of a frog, will sometimes make it contract these muscles The frog must be fixed in a proper frame If repeated observations were made on the muscles thus in action, it might perhaps give some farther insight into the nature of muscular motion

time, into this large field of statical and other experiments, whence we see what great encouragement we have to spur us on in these pursuits, since the wonderful works of the great Author of nature are so fruitful in furnishing us, from its inexhaustible fund, with fresh matter for our researches, and thence with the inexpressible delight, of new and farther motives to adore and praise our all-glorious Maker in his works

*EXPERIMENT XI**

1 As to the force with which the blood is impelled from the right ventricle of the heart into the pulmonary artery, it seems impracticable to attempt the finding of it, by fixing a tube to that artery, in the same manner as to the carotid and crural arteries of living animals, because the animal must needs die while it is doing

2 The area of the transverse section of the pulmonary artery being in one part, before it divaricates into branches, of the same dimension with the orifice of the *aorta*, the velocity of the blood in that part may be accounted the same as in the orifice of the *aorta* But tho' the quantities and velocities of the blood, in passing out of both ventricles, be the same, yet it does not thence follow, that their expulsive forces must be both the same for if the blood in passing into the pulmonary artery, finds less resistance from the preceding blood, than the blood does in entering into the *aorta*, then a less force will expel it out of the right ventricle with equal velocity, and accordingly, as there is not so much force required to drive the blood thro' the lungs, as thro' the rest of the whole body, so we may observe, that the substance of the muscle of the right ventricle has not near the thickness of that of the left The following experiments and observations may give us some light into this matter, *viz*

3 I fixed a glass tube to the pulmonary artery of a calf's lungs, and then thro' a tunnel† poured warm water into it, then with a large pair of bellows fixed to the wind-pipe, I alternately dilated the lungs, to try if by that means the water would pass into the pulmonary vein but I soon found myself disappointed, for the water flowed so freely from the capillary arteries thro' the tunicles of the vesicles, into the vesicles themselves, and thence into the *bronchiae*, as to flow plentifully thro' the wind-pipe, when it hung down in a depending posture At first I suspected that the force of the water, which was four feet high, in the tube affixed to the artery, might have burst the thin blood-vessels, but I found it the same in several trials, on the fresh warm lungs of sheep, oxen, and calves, even when the perpendicular height of the water of the tube was less than a foot, and doubtless the force with which the blood is thrown into the lungs by the right ventricle of the heart, is greater than this

4 And that so small a force of water could not burst the blood-vessels, I was assured by the following experiment, *viz* I dissolved 4 ounces of nitre

*Experiment X has been omitted from the present printing —F A W 1940
†Funnel —F A W, 1940

in a pint of hot water, into which water there flowed from the cut throat of a calf, a quart and a quarter of a pint of blood, which was kept in a diluted state by the nitrated water. Having then fixed a tube which was 2 feet long, to the pulmonary artery of the above-mentioned calf's lungs, I poured gradually into the tube, of the nitrated blood, as much as the artery and its ramifications would contain, which was near a quart, none passing, that I could perceive, into the pulmonary vein. The lungs were much dilated, and looked very red: but notwithstanding the perpendicular height of the blood in the tube was 2 feet, yet no blood passed through the tunicles of the vesicles, into the vesicles and *bronchiae*, for when the wind-pipe was held downwards, nothing flowed out but a white froth, a plain proof that when the water was less than a foot perpendicular in height, in the foregoing experiments, it did not forcibly break through the blood-vessels, but must pass through pores which were too fine for the globules of nitrated blood to pass: those pores being perhaps something larger in the lungs of a dead animal, than when alive, for upon death all the fibres of the body are relaxed. When I cut a slash into the substance of the lungs, the nitrated blood freely flowed out.

5. And that the capillary arteries were not burst by the force of the water, seems farther probable from hence, *viz.* I fixed a tube 5 feet long to the pulmonary vein of a hog's lung's and poured in warm water, which neither flowed into the pulmonary arteries, nor among the *bronchiae*, an argument that this force did not burst the veins, which some anatomists say, have no valves in them.

6. When I fixed the same tube to the wind-pipe of those lungs, and poured in water, it passed through the *bronchiae*, and ran out of the orifice of the pulmonary artery, but not above one-fifth so fast, as when its course was the reverse, *viz.* from the pulmonary artery to the *bronchiae*, in which case it run at the rate of a pint in a minute. Yet when air was blown into the cavity of the lungs, thro' the wind-pipe, none passed thence, either into the pulmonary artery or vein.

7. Another time I tried also whether the thin serum of a hog's blood would pass from the pulmonary arteries, thro' the corresponding veins of the lungs, of the same hog, which lungs were kept warm in water; the serum passed most freely thro' into the *bronchiae*, but not into the veins.

1749

JEAN-BAPTISTE DE SÉNAC

TREATMENT OF "REBELLIOUS PALPITATION" WITH QUININE

JEAN-BAPTISTE DE SÉNAC

(Courtesy American Heart Association)

JEAN-BAPTISTE DE SÉNAC

(1693-1770)

"Theory reduced to consequences drawn from facts alone, is the light of practice"

—Sénac, in preface to *Le Traité de la Structure du Cœur* (1749), quoted by Renouard

JEAN-BAPTISTE DE SÉNAC was born in 1693 in the district of Lombez in Gascony, France From his birth to the date of publication of his first work in 1724 nothing definite about him is known, but it is said that at first, encouraged by his parents, he decided to study for the ministry Later he changed his studies to medicine

There is no definite knowledge as to where or when Sénac studied medicine Degris wrote that Eloy, in his "Biographical Dictionary," said that Sénac received his Bachelor's degree from the Faculté de Paris but Degris thought that this was an error, since Hahn could not find Sénac's name in the "Commentaries of the Faculty" or in Baron's list of recipients of the Bachelor's or Doctor's degree of the Faculty

Sénac, it is known, accepted the appointment of consulting physician to the King in 1738 and on his first publication he signed himself as a member of the faculty of the University of Montpellier, which Noé Legrand called the oldest university in Europe

According to Degris, it is believed that Sénac came to Paris when he was about thirty years of age In Paris he published his first work in 1724, a translation of Heister's "Anatomy"[1] In 1724 and 1725 he communicated before the Royal Academy of Sciences two memoirs "On the Organs of Respiration" and "On Drowning" In 1724 he became an associate member of this academy

In 1727 he published an appreciative account showing the different methods of the lithotomists entitled "Discours sur la méthode de France et sur celle de Rau Touchant l'Opération de la Taille" Sénac published many more interesting contributions, but none is as famous as his "Traité de la Structure du Coeur" (1749), which we shall later discuss in more detail

Sénac moved to Versailles in 1733 where he became physician to the Royal House of Saint-Cyr and to the Royal Hospital of Versailles

In 1745, Sénac cured the great French general, Maurice, Count de Saxe (1693-1750), of a serious disease and later accompanied him on his campaigns In 1751, after the death of Maurice, who was given Turenne's title of "Marshal general of the King's armies and camps," Sénac became chief physician to the Duke of Orleans and in 1752, after the death of Chicoyneau, he became chief physician to Louis XV He treated the dauphin during his illness from smallpox in 1752, and again during the young man's fatal illness from tuberculosis in 1765 In addition to the members of the court he numbered among his patients Madame de Pompadour

In 1760 and again in 1761, Voltaire addressed two letters to Sénac The first supported Sénac's memoir on a certain contagious disease which had devastated the country around Ferney, and the second was a missive of thanks to the "chief physician" for his good work in aiding some people who lived close to a contaminated marsh

Louis XV appointed Sénac counsellor of state and superintendent of the mineral waters and medicinals of the kingdom

Sénac died on December 20, 1770, at the age of seventy-seven years He was survived by two sons, one of whom, Gabriel Sénac de Meilhon (1736-1803), was a writer and was invited to Russia in 1792 by the Empress Catherine II, and the other of whom was a superintendent of agricultural revenues

Sénac's fame in cardiology rests, of course, on his important work first published in 1749 in two volumes, "Traité de la Structure du Coeur, de son Action et de ses Maladies" A second edition of this valuable work appeared in 1777, seven years after the author's death

In his original work, which greatly surpassed studies of the heart made by his predecessors, Sénac made many noteworthy observations Among these are his descriptions in detail of the structure of the heart and the direction in placement of its fibers He discussed the transfusion of blood He noted the increase in incidence of cardiac disease with the increase in age and considered dilatation to be the most common of all cardiac conditions Sénac related pericarditis to inflammation of the lungs and mediastinum He acknowledged that hydrothorax played a conspicuous part in failure of circulation He was the first physician to use quinine for palpitation, and it is this classic description which we are republishing herein in translation According to Long, the germ of the modern idea of septicemia is seen in Sénac's conception of the condition as caused by pus which flowed back into the blood from the loci of external ulcers

Sénac regarded with skepticism the remarks of early writers on "hairy" hearts, and on stones and worms in the heart He believed that polyp of the heart was a formation occurring at the time of death Senac entertained a curious idea about the remote cause of movements of the heart He thought these motions were transmitted by an animal spirit situated in the brain and spinal marrow He thought of this spirit as an extremely elastic fluid, which the impression of the blood on the delicate tissue of the parietes of the heart and the columns of the ventricles put into action

TRAITÉ
DE
LA STRUCTURE DU CŒUR,
DE SON ACTION, ET DE SES MALADIES.

Par M. SENAC, Médecin Consultant du Roy.

Multum egerunt qui ante nos fuerunt, multum etiam adhuc restat operis, multumque restabit, nec ulli nato post mille sæcula præcludetur occasio aliquid adjiciendi. Ann. Seneca.

TOME PREMIER.

A PARIS,

Chez JACQUES VINCENT, rue & vis-à-vis l'Eglise de S. Severin, à l'Ange.

MDCCXLIX.

AVEC APPROBATION ET PRIVILEGE DU ROY.

OPERATION OF STOMACHIC REMEDIES IN PALPITATION*

[Including the use of quinine in rebellious palpitations]

THE above are not the sole resources of Medicine against palpitation, aid has been found in various remedies of which the properties are very different These remedies are the stomachics, the cordials and the sedatives The stomachic remedies have appeared to various physicians as a resource against palpitations, for it is often in the stomach that their cause resides, if they do not arise from this as an immediate cause, there is in many cases an occasional cause which sets the other in motion The ancients, attentive to sensible effects, have accused *flatulence* Pisanus, Higmer and Bartholin have had the same idea It is true that if the first instrument of digestion is inflated by the action of the air that it contains, it will produce the same effects as if it were full of aliment, and when it cannot empty itself it will agitate its viscera and its nerves and may excite palpitation, as Widelius has judiciously remarked The majority, he says, of those who are subject to palpitations are *hypochondriacs,* the functions of their stomachs are deranged, and this derangement troubles the action of the heart

The derangements of the viscera are thus an object that one must not lose sight of in the treatment of palpitations, it excites them often when it is overloaded, thus regularity is one of the necessary conditions to avoid the agitation of the heart or to calm it If it comes from a vice of that organ, sobriety is not less essential, excess adds to this vice a new irritation, the viscera of the lower abdomen being overloaded, are too much pressed on by the mass of the full stomach, and these push the blood with more force and give stimuli to the nerves which may trouble the movements of the heart

When patients are put on a regime one must facilitate digestion if this is possible, such a resource will be the more necessary if the palpitations are excited by the viscera, or if they are occasioned by the derangement of its functions, when one must first of all attempt to prevent food from staying too long in the stomach, and see that food of poor quality be not taken

Bitter extracts and other similar remedies aid the stomach to empty itself, they act on the membranes to stimulate and urge them, they have

*Sénac, J B *Traite de la structure du coeur, de son action et de ses maladies,* Paris, Chez Jacques Vincent 1749, Tome Second, XVIII p 524 Translated by Maurice N Walsh, M D, Mayo Clinic

some of the qualities of the bile, which they replace when it does not flow easily or has lost its qualities, but their operation carries with it some disadvantages, they are warming and thus it is necessary to avoid or to moderate their usage in bodies which are too sensitive and susceptible to impressions of heat The usage of carminatives necessitates the same management, these are not remedies that one can neglect as they have produced happy results according to Widelius and other physicians

If aliments degenerate in the stomach the bad qualities that they produce reduce to two they become acid or rancid It is to absorb the bitter and to correct rancid oils that physicians have resource to earthy absorbents as remedies that may calm palpitations The efficacy of these remedies is recognized by all observers in the malady called *Soda*, that is to say in that derangement of the stomach which sends into the esophagus a burning heat, or a kind of caustic liquor which appears to leave impressions of fire in its passage

In general, remedies which facilitate digestion have been regarded as remedies for palpitation of the heart Widelius has prescribed the elixir de propriété (simple elixir ?), Marcatus recommends Rhapontic as a proven remedy, he gives an infusion of it in wine with Panax, round Aristolochus, Greek Fennel Rivière has given his approval to this remedy which in truth acts at the same time as a purgative and appropriate to hysterical affections

Of all the stomachic remedies the one whose effects have appeared to me the most constant and the most prompt in many cases, is Quinine mixed with a little rhubarb Long and rebellious palpitations have ceded to this febrifuge, seconded with a light purgative

One must place among the stomachic remedies the cordials which have been regarded as effective remedies against palpitation

1755

ALBRECHT VON HALLER

DESCRIPTION OF CALCIFICATION OF THE HEART AND PERICARDIUM

33 Der Grosse Haller

v. n Freudenberger

WIESE, Die Bildnisse Albrecht von Hallers

ALBRECHT VON HALLER

Portrait by Sigmund Freudenberger

(Courtesy Charles C Thomas)

ALBRECHT VON HALLER

(1708-1777)

"The Prince of Physiologists"

—Pettigrew

ALBRECHT VON HALLER was born in October, 1708, at Bern, Switzerland. His father was a prominent lawyer and Albrecht was his fourth and youngest son. Young Haller showed a large capacity for knowledge in early childhood. At the age of four he expounded the Scriptures to the servants, at eight years of age, he had written 2,000 brief biographies, and by the time he was ten years old he had written a brief lexicon of Greek, Hebrew, and Chaldean.

Haller continued to astound his family with his intellectual achievements, for at the age of twelve he began to write verse. When he was either fourteen or fifteen, we are told, he wrote an epic poem of 4,000 stanzas on the "Origin of the Swiss Union of States." At this time he also translated Ovid, Horace, and Virgil.

In 1723 Haller matriculated at the University of Tubingen. Two years later he traveled to Holland, where he studied under the renowned Boerhaave. He later wrote a commentary on the works of his great teacher. At the age of eighteen, Haller received his medical degree from the University of Leyden. In his dissertation he disproved Coschwitz' supposed discovery of a new salivary duct. Haller proved this anatomic structure to be a blood vessel.

After receiving his degree, Haller traveled to France and England where he studied for a time before returning to Switzerland. Then he went to his native country, studied mathematics under Bernoulli in Basel and later returned to Bern to practice medicine. In 1734, when the Republic of Bern established an amphitheater, Haller was invited to give demonstrations in anatomy.

With the founding of the University of Gottingen in 1736, Haller was asked by George II, King of England, who was also Elector of Hanover and Brunswick, to fill the chair of anatomy, surgery, and botany. Haller accepted and remained in Göttingen for seventeen years. At Gottingen he founded the Anatomical Museum and Laboratory, the Botanical School and Garden and the Department of Obstetrics. He also founded there a journal entitled "Gottinger gelehrten Anzeiger," to which he contributed more than 12,000 book reviews. During this time, also, he received attractive offers from Oxford, Leyden, and Berlin, but refused them all. In 1752 the Emperor Joseph conferred a baronetcy on Haller. This he did not accept.

Haller returned to Bern in 1753, where he remained until his death in 1777. In his last days he suffered from an inflammatory condition of the bladder so painful that he could be relieved only by large doses of opium.

The most productive period of Haller's literary career began in Gottingen. He was perhaps, the most prodigious and versatile writer of all times. According to Garrison, while Haller was in Göttingen he wrote some 13,000 scientific papers. We have mentioned his 12,000 book reviews contributed to the journal he had founded. At Göttingen, also, Haller began his classic experiments on nerve sensibility and

muscular irritability which were to be published in 1757 And at Gottingen he laid the foundation for his huge and most authoritative work in physiology, "Elementa Physiologiae corporis humani" This was published in nine volumes at Lausanne from 1759 to 1769

It is impossible in this brief account even to mention all of Haller's contributions to medicine As far as we know, he was the first to describe calcification of the pericardium This description we are including herein

Before Haller published his great work on physiology, he contributed several monographs on physiology and its relationship to embryology, circulation, reproduction, formation of bone and irritability

According to Hemmeter, the principal contributions of Haller to anatomy were (1) a demonstration that the so called salivary duct was a vein, (2) an investigation of the respiratory muscles and an exhaustive description of the diaphragm, with an interpretation of the intercostal muscles as elevators of the ribs, (3) a demonstration of the uterine musculature, (4) a demonstration of the coni vasculosi, (5) a correct description of the musculature of the heart and an accurate description of the pericardium and the valves in the veins, (6) a description of a number of hitherto unknown arteries, (7) recognition of the higher situation of the bladder above the pubes in children, (8) description of the omentum and (9) demonstration of the tela cellulosa as a connective tissue substance

To cardiac physiology Haller made many contributions He stressed the changes of the heart occurring during contraction and he was familiar with the influence of gravity and respiratory aspiration of the thorax on the circulation of the veins He also succeeded in demonstrating the automatism of the heart

Haller gained universal admiration for his several works on botanical subjects and in his "Historia stirpium indigenarum Helvetiae" he described 2486 species of plants He holds an important place in the history of German literature, his poetry having been regarded as outstanding His well-known poem, "The Alps," was published in English in 1729 Twenty-two editions of it were published in German and it was also translated into French, Italian, and Latin He also contributed to history his splendid biography, "Life of Alfred the Great"

By all means, mention should be made of his numerous bibliographic undertakings Haller had made it a custom to write a concise summary of the books he read His "Bibliothecae," comprising his bibliographies on anatomy, botany, surgery, and medicine, compiled from his lifetime of reading, include 52,000 works! According to Fulton, next to the "Index Catalogue of the Surgeon General's Office" of the United States Army, this collection represents the greatest compilation of medical titles ever assembled Not only is it an index to the world's medical literature up to and including Haller's era, it is also a compendium in which each author's biographic data are given, a summary of the work is presented, critical comments on the author's views are offered, and other considerations that Haller thought of interest are included

Haller was married three times His first two wives died and by his third wife he had eleven children Of these, four sons and four daughters survived him Among the few honors that he chose to accept were membership in the Academy of Sciences of Paris and fellowship in the Royal Society of London Gustavus III, King of Sweden, also made him a knight of the Polar Star

ALBERTI HALLERI

PRÆSIDIS S. REG. SC GOTTING.

OPUSCULA PATHOLOGICA

Partim recusa partim inedita.

QUIBUS

SECTIONES CADAVERUM

MORBOSORUM

Potissimum continentur.

ACCEDUNT EXPERIMENTA

DE RESPIRATIONE,

QUARTA PARTE AUCTA.

LAUSANNÆ,

Sumpt MARCI-MICH. BOUSQUET & Soc.

MDCCLV.

DESCRIPTION OF CALCIFICATION OF THE HEART*

But the following unusual disease is worthy to be remembered, from it a most admirable young person died not long since. The mother of the boy I perceived on medical examination eight years previously to be subject to palpitation of the heart, the youth now fell into the same illness. He himself, on the day of his death, was without the pulse which you feel in the wrist, however I *found the carotids to be violently throbbing*. [The patient] chilling, then drenched with sweat, I reluctantly gave a bad prognosis.

Shortly after death we opened the body. The pericardium of the heart and the pleura of the lungs were everywhere attached, and all over the surface of the pericardium were white hard masses, some firm and some filled with white *material like pus*. These hard swellings were totally and indissolubly united to the pericardium by bands. The semi-stone-like inferior part of the right ventricle was strongly adherent to the pericardium by a mass of tophaceous calculi like fine sand. The sinus between the two membranes of the aortic valves was hard and in part stony. In the aortic valves between the membranes there was a strange material, in fact the tendons which held back these valves were even found to be fleshy, and with variable bony scales.

But a special disorder was concealed in the valves of the pulmonary vein. These were all excessively hard and solid, they were completely filled with stony material, to the extent that the fibres grated whenever they were cut. Even the tissue of the pulmonary sinus was formed of stony material. Neither the heart nor the great vessels exceeded the usual size.

The patient's age, twenty years, increases the rarity of the disease. The heart of this youth was not stopped up, neither was it satisfactorily open, it lacked alternate rest, without which no heart can live. Now the left ventricle received blood with great difficulty from its sinus and by its contraction sent it through the rigid opening of the osseous mitral valves to its sinus. Thus from the aorta in like manner the blood could return between the inexplicably rigid aortic valves into the heart. Whence, with the heart perpetually stimulated, it palpitated uninterruptedly, and since it could not send enough blood to the brain, in this manner a stupor was produced, like that which befalls with loss of blood from venesection or from wounds.

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*Von Haller, Albrecht. *Opuscula Pathologica,* Lausannae: Bousquet et soc., 1755, p. 135. Translated by Maurice N. Walsh, M.D., Mayo Clinic.

1761

JOHN BAPTIST MORGAGNI

DESCRIPTIONS OF MITRAL STENOSIS, HEART BLOCK, CALCAREOUS STENOSIS OF THE AORTIC VALVE WITH REGURGITATION, CORONARY SCLEROSIS, AND ANEURYSM OF THE AORTA

JOHN BAPTIST MORGAGNI

(Courtesy Charles C Thomas)

JOHN BAPTIST MORGAGNI

(1682-1771)

"Those who have dissected or inspected many (bodies), have at least learned to doubt, when others, who are ignorant of anatomy and do not take the trouble to attend to it, are in no doubt at all"

—Morgagni, letter 16, article 25, of *De sedibus et causis morborum*, after Adams

MORGAGNI was born on February 25 near Bologna at Forli, at that time an important Italian town He matriculated at the University of Bologna, where he studied under the faculties of philosophy and medicine He was graduated from Bologna in 1701, with high honors from both faculties

Soon after graduation Morgagni accepted at Bologna the position of prosector in anatomy under the famous teacher, Antonio Maria Valsalva, a pupil of Malpighi Morgagni assisted Valsalva in the preparation of the latter's famous work on the anatomy and diseases of the ear [1] This work was published in 1704

Morgagni esteemed Valsalva and later edited the anatomic writings of his former teacher Morgagni's editing of Valsalva's work was supplemented by some of his own observations, and he later added a memoir to the life of Valsalva (1740)

When Valsalva resigned his position to accept an offer at Parma, Morgagni succeeded him at Bologna as demonstrator in anatomy In 1706 Morgagni became president of the Academia Inquietorum of Bologna At this time he published in six parts his first notable work, "Adversaria anatomica," comprising the substance of his communications to the Academia Therein are contained some of his independent discoveries, more especially, as Nicholls points out, "in connection with the muscles of the hyoid bone, the uvula and the larynx"

After some time, Morgagni resigned his position at Bologna and went to Padua and Venice, at which universities he continued his anatomic studies for the space of two to three years He then moved to his native town, Forli, and began the practice of medicine Although Morgagni was successful at the physician's art he did not enjoy the practical aspects of medical practice, and, therefore, on the death of Domenicho Guglielmini, he accepted a chair of theoretical medicine in the University of Padua made vacant when Antonio Vallisnisei succeeded to Guglielmini's post

Thus began Morgagni's long and uninterrupted academic career, for he taught at Padua until his death fifty-one years later After three years he was appointed by the Venetian Senate to the chair of anatomy at Padua made vacant by the death of Michel Angelo Molinetti This was the most distinguished position on the faculty, and among the renowned anatomists he succeeded were Vesalius, Fallopius, Casserius and Spigelius

Morgagni was popular with his students, and his distinguished friends, according to Pettigrew, included King Charles Emanuel III of Sardinia, and Popes Clement XI, XII, and XIII and Benedict XIV In his own profession he enjoyed an international reputation and was esteemed by Valsalva, Albertini, Lancisi, Verheyen, Heister, Ruysch, Boerhaave, Mead, Sénac, Haller, Meckel, Le Clerc, Fantoni, Nigrisoli, Michelotti, Molinetti, and numerous others

Morgagni was the recipient of scientific honors from all Europe He was elected a member of the Academia Naturae Curiosorum in 1708, the Royal Society of London in 1724, the Academy of Sciences of Paris in 1731, the Imperial Academy of St Petersburg in 1735, and the Academy of Berlin in 1754

Apart from his scientific studies, Morgagni was a man of unusual attainments He wrote on philology, archaeology, literature and history Nicholls mentioned these works as typical Morgagni's letters to Lancisi on the "Manner of Cleopatra's death," "Commentaries" on Celsus and Sammonicus, and notes on Varro, Alpinus, Vegetius, Columella, and Vitruvius, as well as archaeologic papers on the districts around Ravenna and Forli

It was not until 1761, when Morgagni was seventy-nine years old that his monumental work, "De sedibus et causis morborum per anatomen indagatis libri quinque," was published This work has immortalized Morgagni as the father of pathologic anatomy, chiefly because the records of post-mortem descriptions are correlated with clinical observations on a grand scale in addition to the fact that he was the first to describe many diseases Before Morgagni's work was published, the standard printed work on the subject was the "Sepulchretum" of Theophilus Bonetus, published at Geneva three years before Morgagni himself was born This work contained what was known about morbid anatomy up to the date of the publication The more Morgagni studied the "Sepulchretum," the less satisfied with it he became and finally he concluded to supplement it with his own observations

As was the custom of the time, Morgagni wrote his observations in the form of letters to a friend, a procedure that resulted in the production of seventy letters The letters were then returned to Morgagni, were revised and published in a huge work of five books

The five books of "De sedibus et causis morborum" deal with (1) diseases of the head, (2) diseases of the thorax, (3) diseases of the abdomen, (4) diseases of a general nature, and diseases requiring surgical treatment and (5) such things as were added to the other four books

The work is based on the results of 640 post mortem examinations, generally occurring in his own experience but occasionally taken from the unpublished notes of Valsalva and Albertini

Morgagni devoted several letters of "De sedibus" to a study of the diseased heart, in which he accurately described the principal cardiac lesions which he found after the death of his patients We have chosen to reproduce his classic descriptions of (1) mitral stenosis, (2) heart block, (3) calcareous stenosis of the aortic valve with regurgitation, (4) coronary sclerosis, and (5) aneurysm of the aorta

As we have noted in our study of Heberden, Morgagni also included an early description of angina pectoris, which he noted in 1707, and published in his "De sedibus" in 1761 (vol 1, p 282) Morgagni observed that certain disorders, such as asthma and dyspnea, formerly considered to be the result of pulmonary disease, also might be caused by diseases of the heart He suggested the possible relationship, as did many of his predecessors, of syphilis to aneurysm He also described rupture of the heart, but did not mention the cause of this condition He also described vegetative endocarditis

Nicholls called attention to the fact that Morgagni "came far short of establishing a complete system of Morbid Anatomy" Morgagni could not entirely shake himself free from the erroneous conceptions of disease prevalent in his day Although the microscope was in use before his time, as we have shown, he does not appear to have used it in his investigations However, by basing his views on personal observation and making an effort to harmonize clinical manifestations of disease with the morbid appearances of diseases, Morgagni established a firm basis for those great principles that underlie modern methods of scientific research

Morgagni's death occurred on December 5, 1771, when he was at the advanced age of ninety The citizens of Forli erected a bust to him in the principal palace of the town in 1763 In Dryden's "Oedipus," the play he wrote with Nathaniel Lee, are found the lines which provide an excellent strophe on the last years of Morgagni

"Of no distemper, of no blast he died,
But fell like autumn-fruit that mellowed long,
Ev'n wondered at because he dropp'd no sooner
Fate seemed to wind him up for fourscore years
Yet freely ran he on ten winter's more,
'Till, like a clock worn out with eating time,
The wheels of weary life at last stood still"

(Quoted by Pettigrew)

THE
SEATS and CAUSES
OF
DISEASES
INVESTIGATED BY ANATOMY,
IN FIVE BOOKS,

CONTAINING

A Great Variety of DISSECTIONS, with REMARKS.

TO WHICH ARE ADDED

Very ACCURATE and COPIOUS INDEXES of the PRINCIPAL THINGS and NAMES therein contained

TRANSLATED from the LATIN of

JOHN BAPTIST MORGAGNI,
Chief Professor of Anatomy, and President of the University at PADUA,

By BENJAMIN ALEXANDER, M. D.

IN THREE VOLUMES

VOL. I.

LONDON,
Printed for A MILLAR, and T CADELL, his Successor, in the Strand, and JOHNSON and PAYNE, in Pater-noster Row
MDCCLXIX.

closely, out of the body. The blood was also black and grumous in the ventricles of the heart, yet not in very great quantity. The right valvula mitralis was white, and in like manner some of the semilunar valves: the former were much harder than usual, and the latter a little so: but in both mitral and semilunar, the membranous nature had degenerated almost into the nature of a ligament. In the middle and posterior surface of the heart, a kind of little membrane protruded, of a white colour, and look'd like the remains of an hydatid. On the right auricle externally, also, were some white spots. But the aorta and other vessels, as far as I could see, were according to their natural appearances.

27. It does not escape me, that you may think this man's death is rather to be imputed to a syncope, than to an apoplexy, as well when you consider the celerity of it, as the sudden coldness of the body at that hot season, and in bed, or even the appearances observ'd in the heart. But, to begin with these last, and to compare them with the quantity of blood found within the cranium, we certainly find greater marks of disorder in the hearts of those, who had not the slightest appearance of fainting, and much less the most violent syncope. And Galen has taught us, that an apoplexy may be form'd from such an abundance of blood within the cranium, though I have never seen any other case of the kind that I remember. Galen's (*n*) words are, "By this means apoplexies are brought on, to wit, by much blood rushing tumultuously into the principle of animation." Nor did Petrus Salius (*o*), who first wrote a separate chapter on the sanguineous apoplexy, as I have mention'd in the former letter (*p*), imagine, that the disorder was, in general, brought about by other means, in the cerebrum, than from "too great a repletion of the veins, arteries, and sinusses, with blood, for which reason a very great stricture is brought upon the brain, whereby, not only the free passage of the spirits is prevented, but they are even choak'd up, and suffocated thereby, so that sense and motion are suddenly lost, and the intelligent faculties are suspended: that is, a true and exact apoplexy succeeds." And, indeed, such a quantity of blood could not be collected in all the vessels within the cranium, but that the soft substance of the cerebrum and cerebellum must be violently compressed, both from within and from without, the small vessels also, which escape the senses, being streighten'd, the circulation of the blood is intercepted, and consequently the secretion of spirits, which cannot happen without it, is prevented: for as there is no vacuity in the cranium, and the bones of it are incapable of giving way, the whole force of the pressure must be expended on the brain. These things cannot happen to the cerebellum, as it seems, and as I have already said (*q*), without a sudden suffocation of respiration and the motion of the heart, being the consequences thereof, that is, with-

(*n*) Vid. apud Salium, de Affect. partic. c. 2.
(*o*) Ibid.
(*p*) n. 1.
(*q*) Epist. 2. n. 24.

out sudden death and this death if you would rather have it so, from a syncope, but a syncope that would proceed from the head and not from the heart, or if from the heart also, on account of the appearances spoken of, yet, at least, certainly more from the head, than from the heart. But wherever there is a syncope, from whencesoever it proceeds there is no reason to wonder at the sudden coldness of the body though I should rather suppose, that it was only a diminution of warmth, which seemed to a woman who was herself warm, to be cold. And, as far as I can judge, you would not err much, if you should call that kind of syncope, which Herophilus (*i*) seems to have particularly pointed out an apoplexy from the cerebellum for he says, "When sudden death happens without any manifest cause, then it is owing to a palsy of the heart" for what an apoplexy from the cerebrum does in other parts of the body, the same an apoplexy from the cerebellum does in the heart but in an apoplexy which proceeds both from cerebrum and cerebellum at the same time, motion is destroy'd in all parts of the body at once. And such, I think, was the case under consideration since there was evidently a material cause of compression upon them both, nor did I find that any of those symptoms had preceded, which are generally antecedent to a syncope from the heart or any of its nearest vessels.

had not wish'd for me to be their companion, in the cure of this refractory disorder, less than the patient himself, made no scruple to pronounce, that it arose from the irritation of the hypochondria. And indeed, as you have it also in this section of the Sepulchretum (*m*), there is extant in Galen a history of a certain grammarian, "who, having abstain'd too long from food, became epileptic, from no other cause than bile." And examples are very common of adults (*n*), not only of children (*o*), who have been troubl'd with epilepsies, from worms harbour'd in the intestines. And to this purpose also is that observation of Spigelius (*p*), on a whelp thus kill'd by worms, not very unlike to which, is that formerly written by me to Vallisneri, and by him publish'd (*q*). And you know that this disorder often arises, also, from other viscera of the belly being diseas'd, which the section, that I have already quoted, confirms (*r*).

But although that kind of cure was applied to my fellow-citizen Poggi, with my assent, which was proper to open, cleanse, and relax, the hypochondria, yet, nevertheless, the accessions still return'd frequently, so that we now began to fear, lest the head itself had also contracted the injury, especially as, upon a very quick turn of the head, the epileptic insults recurr'd, and left a sense of weight with stupidness in the head, and frequently some blood came, together with the mucus, from the nose. Wherefore, as in the beginning, they had already drawn blood once and again from the arm, nor had omitted to give such things as are generally of use to the head, I persuaded them to let blood be taken away from those veins, which lie about the anus also, and that several things should be given internally, which are recommended as extremely proper against this disease, by the most excellent physicians. These remedies, however, were of no advantage, but the bleeding, whether it reliev'd the head, or rather those viscera which are serv'd with blood by the vena portarum, was so far of advantage, that for a short time the paroxysms were quiet. When, therefore, they return'd again more frequently, it was of use to make the patient sit up, sometimes to rub the lower limbs, and sometimes to tie them alternately with bandages thrown round about, and sometimes to fix cupping-glasses without scarification, and presently to vex the patient by taking them off, for thus he seemed to have a longer intermission from his paroxysms. And I was even assur'd, that when they sometimes attack'd him much more often, the spirit of salt ammoniac, applied to the nostrils, had driven them away as they were coming on, or, even when they were already, in a manner begun, had suppress'd them, although the patient was entirely without the power of smelling. They were, for the most part, very short, but by no means slight. For distortions of the eyes, agitations of the limbs, and a suspension of all the senses, always accompanied the

(*m*) Sect 12 in schol ad obs 19
(*n*) Ibid schol ad obs 41 (*o*) Obs ead § 2 & schol ad obs 15 in additam
(*p*) Ibid obs 41 § 1 (*q*) Consideraz in a la gener de Vermi
(*r*) Obs 39 cum schol

attack oftentimes there was a strangulation, and that sometimes join'd together with a stertor and even now and then an involuntary efflux of urine attended But he was exceedingly bad that day on which the solstice happen'd, and in like manner that on which the eclipse of the sun happen'd

And though you may suppose this might be by chance, yet you cannot suppose it merely accidental, that when the quantity of urine was either naturally or artificially encreas'd (s), the epileptic paroxysms not only became not slighter, but were even very frequently exasperated For we were oblig'd to have regard to this excretion sometimes when a sudden difficulty of breathing rous'd the patient, as he was beginning to sleep and compell'd him to sit up, which symptom, doubtless gave us some suspicion of a dropsy of the thorax, and the more so, because the patient told us that his right leg had, for a long time past, been accustom'd to swell a little with water, and that even then which, when he told us, we examin'd into, the swelling was ascending up the thigh But it was easy to encrease the quantity of urine, by obvious and innocent remedies and therefore to diminish the tumour, and that suspicion, which was afterwards entirely remov'd, but not so the force of the attacks which, from the encreas'd afflux of urine, and that of itself, sometimes opaque, and blackish was so far from being weaken'd, or diminish'd, that even on the contrary, as I said above, they grew stronger and stronger When these things, and others, which for the sake of my promis'd brevity I pass over were of no effect against the inroads of this disease, and even such as had been sometimes useful to retard or suppress them as I said above, were now of no advantage, as they did not continue to afford these effects, there was one thing, however,

mended to dissolve, and promote, the circulation of the blood, in vain, upon giving the opium again, the quiet nights again return'd, and diminish'd that inequality of the pulse and, by the continu'd use of opium every night, it was entirely remov'd, and even the former rarity was diminish'd

But, perhaps, you will suspect, whether the rarity of the pulse be, in fact, a very uncommon symptom, to remain after an epilepsy, in hypochondriac patients, when you shall have compar'd this observation of mine with that of the celebrated Gerbezius (*t*) which describes the pulse of a strong hypochondriac man, "who was now and then subject to slight epileptic paroxysms," even when he was in health, "as being so very slow, that before the subsequent pulsation follow'd that which went before, three pulsations would certainly have pass'd in another healthy person " But to return to my subject, after that no fit had now returned for thirteen days, and the use of opium was intermitted, the first night indeed was not bad, but the following ones, by reason of the continual watching, and restlessness, and at length by reason of that difficulty of breathing, which I spoke of above, were exceedingly troublesome, so that we were oblig'd to have recourse again to opium, in order to procure quiet nights, which nothing but opium would procure And, to comprehend all in a few words, that the attacks of the disease, from being very frequent, as they had happen'd every day, in the month of June, had been so far reduc'd in their number, that but one happen'd in July, one in August, nor more in September, and after that none in the two next months at least, and upwards, till I departed to teach medicine publicly, we judg'd was owing to the use of opium, given opportunely, sometimes every night, sometimes every other night, and at length at the intervals of many nights For by that medicine we were able to appease the tumultuary motions, which arose, and frequently by a very manifest sensation, from the hypochondria, to the thorax, and head, and by this means procure a truce, both for nature and art, and this gave us sufficient time to cleanse and confirm the hypochondriac viscera, which we had determined to do, in the beginning, but in vain attempted, among those first continual tumults, with which the patient was harrass'd and from these viscera alone, and not from water being redundant in the brain, that these sudden commotions arose, this history, or I am much deceiv'd, indeed, evidently shews

(*t*) Eph N C Cent 7 in Append

LETTER THE TWENTY-THIRD

TREATS OF PALPITATION AND PAIN OF THE HEART

[Description of Calcareous Stenosis of the Aortic Valve with Regurgitation]

8 A woman, a little younger than that last describ'd, complain'd in the same hospital of a palpitation of the heart sometimes, but always of a difficulty in her breath, which she could not draw but with her neck erect, and still more of so great a streightness and anxiety at her heart that very often she seem'd just at the point of death. Some suppos'd her to labour under a dropsy of the pericardium. Her pulse never was intermitting, but her veins were large. She died at the time that the genital parts of a woman were wanted to finish the public demonstrations of the year 1731, a little before the middle of March.

The thorax and belly being open'd, some quantity of water was found in both cavities, but there was no dropsy of the pericardium. The valves of the aorta were indurated, and one of them even bony. The trunk of the artery itself shew'd, up and down on its internal surface, either something bony, or something verging to the nature of a bone, so that the part of the artery which went through the belly, and which I dissected after demonstration of the genital parts, was in the same state. Nor did I find it bony only at the side of the inferior mesenteric artery, and in other places, but even at the very division of it into the iliacs; and in several places it was unequal, and here and there of a whitish colour, as it generally is when it begins to become bony.

9 It certainly cannot be denied that the aorta, in the state I have describ'd it, must resist the blood as it is driven by the heart, and, for that reason, be able to create a palpitation, a difficulty of breathing, and that sense of streightness with which the woman was tormented. But at the same time it is necessary to declare why, out of so great a number of persons in whom there was an aorta of this kind, as I have already written to you, and shall write hereafter, many of them, certainly, did not labour under these disorders at all, or, at least, not so vehemently. And in order to do this, other circumstances, without doubt, must be added to the disorder of the aorta, which did exist in this woman, and did not exist in the others, as for instance, a different fault in the organs, or a different constitution of the blood, different quantity, and other things of a similar kind, besides that we may not seem to be always bringing in the more exquisite sense of the nerves, and convulsions. So in an observation of Verdriesius, already pointed out (a), after a violent palpitation of the heart and a very great asthma, the aorta was found to be internally bony near the heart, but the heart itself was found to be of an extraordinary magnitude [illegible] [illegible]

(a) [illegible]

disorders, had been, in the beginning of them, very much subject to a palpitation of the heart, the celebrated Plancus (*n*) not only found the aorta in many places become bony, but also both the coronaries of the heart, and the heart itself very large, particularly its right auricle, which was the largest and strongest of all and from hence you may easily perceive, that, in consequence of this one disorder, the heart might be more vehemently irritated by the blood being more strongly impell'd into the subjected ventricle, and may the less wonder, if in the observation of Grassius the younger, spoken of already in this work (*o*), it shall perhaps appear, that nothing else could be the cause of the palpitation, but the right auricle being enlarg'd to the double of its usual capacity Finally, not to detain you too long, in a woman of an illustrious family, whose palpitation of the heart was so great, and so constant, as to be heard by those who stood near her, and be discern'd by those who were at some distance, the celebrated Cohausen (*p*) not only saw the aorta entirely callous, but he even saw in the heart itself, not to mention the lungs, scirrhi, and the blood viscid and mucous

Wherefore, in the woman, also, spoken of by me, besides the aorta being here and there bony, or inclining to a bony state, the valves of it are also to be attended to For as one of these was bony, and the others indurated, so being, of consequence, less yielding to the blood, they might encrease the obstacles to its exit, and, on the other hand, not sufficiently prevent its return, when, soon after, repuls'd by the contraction of the great artery, so that, as some portion of it return'd into the left ventricle of the heart, when this ventricle ought to receive the blood that was coming in from the lungs, it would necessarily happen, that the returning portion, as well as the portion which had not been extruded just before, must occupy some part of that space, which, from the design of nature, was entirely due to the blood that was coming in from the lungs Which circumstance, finally, could not but overload both the lungs and the heart, and compel the latter to throw out, every now and then, with a great impetus, the blood that stagnated in it

And these things, which reason seems to lead us to, are confirm'd by more than one observation of the anatomists Thus Vieussens (*q*), in a man and a matron, both of whom had been long troubl'd with a palpitation of the heart, and incapacity of lying down with the head low, join'd with an inequality of the pulse, relates, that the aorta and its valves were found to be bony in the matron, and in the man, that the aorta was very hard, and in a manner cartilaginous, and the valves not only bony, but also with their edges cloven asunder, and the same really become stony, I say really, lest you should, perhaps, imagine, that what was bony should have been taken for being stony in the heart, as it has happen'd sometimes in other

(*n*) Epist de Monstr (*o*) Epist 18 n 4 (*p*) Commerc Litter A 1743 Hebd 21 n 4

(*q*) Traité du Coeur ch 16

places For besides those which will be mention'd below, you will read of similar cases, here, in the Sepulchretum, in observations which are pretty ancient, as that of Gregorius Horstius the elder (r), who found "a calculus concreted from tartar adhering to the membranous substance of the valves" of the right ventricle of the heart, and that of Jo Georgius Greiselius (s), who saw one of those valves of the aorta which we are speaking of "half-consum'd, and lost, and that part where it adher'd to the artery even friable into a kind of sand", and there was "a kind of white particle, like the nail of a little finger," which was the part of the valve that the blood had lately torn away, the heart being in both of them, who died after a palpitation, very much enlarg'd, in the same manner as the left ventricle had been in that man of Vieussens

LETTER THE TWENTY-FOURTH

TREATS OF PRETERNATURAL PULSES

[Description of Coronary Sclerosis]

16 In an old man, who was of a lean habit, and whom I dissected in the month of December of the year 1743, the pulse had been weak and small, but not intermitting, when, on account of an *incarcerated* hernia, as it is call'd, he was brought into the hospital at Padua And notwithstanding it was out of my power certainly to inform myself, whether the pulse had been in that state before this disorder came on, or whether it was rather brought on by this disease, join'd with an inflammation of the intestines, to such a degree, that a speedy death prevented any method of cure being attempted, yet the appearances which I observ'd in many parts of the body, and particularly in the heart itself, and demonstrated to a very crowded circle of students, are of such a nature, that I cannot judge them to be unworthy of being communicated to you

As I examin'd the external surface of the heart, the left coronary artery appear'd to have been chang'd into a bony canal, from its very origin to the extent of many fingers breadth, where it embraces the greater part of the basis And part of that very long branch, also, which it sends down upon the anterior surface of the heart, was already become bony to so great a space, as could be cover'd by three fingers plac'd transversely For which reasons, a passage was open'd on both sides, not through a membranous canal, or one which was made somewhat hard, here and there, by disjoin'd bony lamellae, but through a continued bony tube, which for hardness might with justice have been compar'd with any other hard bone, except that in some places it was less hard, though those were very small, and inconsiderable, and resembl'd the transverse lines form'd by the

(r) Obs 35 (s) Obs 13

knots of a slender reed The heart then being open'd, and some polypous concretions being taken away, although I saw the tubercles of the valves of the great artery much harder than usual, and almost bony, yet I found nothing bony either in them, or in any other valves, or in that artery near the heart But at some interval from the heart, and at the origins of the upper arteries, and from thence downwards quite to the divisions into the iliacs, the internal surface of the aorta was frequently unequal on account of very hard bony laminae, many of which equall'd in bigness the nail of a man's thumb yet I found the thin internal membrane, by which all these ossifications were cover'd, to be hurt only in one place, a thickish kind of humour showing itself there in regard to which, and the seat itself of these laminae, I shall have a more convenient opportunity of speaking hereafter (*x*), and telling you what I observ'd in this man, and in others And bony scales were not wanting, either at the division into the subclavian and carotid arteries, on the right side, or in the iliacs, nor yet in the splenic in particular, in which they were found very thick, quite to the spleen Nevertheless, within the cranium, and in like manner, both in the upper limbs, and in the lower limbs, I observ'd nothing bony in the arteries, although this class of vessels was, in the limbs, more firm and hard than usual, and perhaps even somewhat wider than they generally are and while I was cutting into these arteries, I saw that the blood which remain'd in the crural vessels was not fluid indeed, yet not polypous

LETTER THE TWENTY-SIXTH

TREATS OF SUDDEN DEATH, FROM A DISORDER OF THE SANGUIFEROUS VESSELS, ESPECIALLY THOSE THAT LIE IN THE THORAX

[Description of Aneurysm of the Aorta]

9 A man who had been too much given to the exercise of tennis and the abuse of wine, was, in consequence of both these irregularities, seiz'd with a pain of the right arm, and soon after of the left, join'd with a fever After these there appear'd a tumour on the upper part of the sternum, like a large boil by which appearance some vulgar surgeons being deceiv'd, and either not having at all observ'd, or having neglected, the pulsation, applied such things as are generally us'd to bring these tumours to suppuration, and these applications were of the most violent kind As the tumour still encreas'd, others applied emollient medicines, from which it seem'd to them to be diminish'd, that is, from the fibres being rubb'd with ointments and relax'd, whereas they had been before greatly irritated by the applications But as this circumstance related rather

(*x*) Epist 27 n 22

to the common integuments, than to the tumour itself, or to the coats that were proper thereto, it not only soon recover'd its former magnitude, but even was, plainly, seen to encrease every day. Wherefore, when the patient came into the Hospital of Incurables, at Bologna, which was, I suppose, in the year 1704, it was equal in size to a quince, and what was much worse, it began to exsude blood in one place, so that the man himself was very near having broken through the skin (this being reduc'd to the utmost thinness in that part, and he being quite ignorant of the danger which was at hand) when he began to pull off the bandages, for the sake of showing his disorder. But this circumstance being observ'd, he was prevented going on, and order'd to keep himself still, and to think seriously and piously of his departure from this mortal life, which was very near at hand, and inevitable. And this really happen'd on the day following, from the vast profusion of blood that had been foretold, though not so soon expected by the patient. Nevertheless, he had the presence of mind, immediately as he felt the blood gushing forth, not only to commend himself to God, but to take up with his own hands a bason that lay at his bed-side, and, as if he had been receiving the blood of another person, put it beneath the gaping tumour, while the attendants immediately ran to him as fast as possible, in whose arms he soon after expired.

In examining the body before I dissected it, I saw that there was no longer any tumour, inasmuch as it had subsided after the blood, by which it had been rais'd up externally, had been discharg'd. The skin was there broken through, and the parts that lie beneath it with an aperture, which admitted two fingers at once. The membrana adiposa of the thorax discharged a water during the time of dissection, with which some vessels were also turgid, that were prominent, here and there, upon the surface of the skin in the feet and the legs. In both the cavities of the thorax, also, was a great quantity of water, of a yellowish colour. And there was a large aneurism, into which the anterior part of the curvature of the aorta itself being expanded, had partly consum'd the upper part of the sternum, the extremities of the clavicles, which lie upon it and the neighboring ribs, and partly had made them diseas'd, by bringing on a caries. And where the bones had been consum'd or affected with the caries, there not the least traces of the coats of the artery remain'd: to which, in other places, a thick substance everywhere adher'd internally, resembling a dry and lurid kind of flesh, distinguish'd with some whitish points, and this substance you might easily divide into many membranes, as it were, one lying upon another, quite different in their nature from those coats to which they adher'd, as they were evidently polypous. And these things being accurately attended to, nothing occur'd besides that was worthy of remark.

1761

LEOPOLD AUENBRUGGER

ON PERCUSSION

LEOPOLD AUENBRUGGER

Painted in 1770 by an unknown artist, restored by Kurz von Goldenstein

(Courtesy Annals of Medical History)

JOSEPH LEOPOLD AUENBRUGGER

(1722 1809)

[*Durdles*] *'Now, lookee here You pitch your note, don't you, Mr Jasper?'*

[*Mr Jasper*] *'Yes'*

[*Durdles*] *'So I sound for mine I take my hammer, and I tap I tap, tap, tap Solid! I go on tapping Solid still! Tap again Holloa! Hollow! Tap again, persevering Solid in hollow! Tap, tap, tap, to try it better Solid in hollow, and inside solid, hollow again! There you are! Old 'un crumbled away in stone coffin, in vault!'*

—From *The Mystery of Edwin Drood,* by Charles Dickens

PERCUSSION as a means of testing whether walls were solid or whether they covered hiding places, whether barrels were empty or full and for many other purposes, must have been in use since the beginnings of civilization It is surprising, therefore, that no one before Auenbrugger seems to have thought of using percussion of the human body as a means of diagnosis

Joseph Leopold Auenbrugger was born at Gratz in Stryria, Austria His father was an innkeeper, and the younger Auenbrugger himself was an accomplished musician[1] These two facts had no little significance in his development of percussion The technic he probably learned from tapping wine barrels in his father's inn and his good musical ear helped him greatly in the interpretation of sounds

In due course Auenbrugger studied medicine at the University of Vienna, where he was the pupil of van Swieten who at an earlier date had been a student of Boerhaave at Leyden Van Swieten had been called from Holland to the Austrian capital by the Empress Maria Theresa He became court physician and because of his royal backing he was able to found the great medical school at Vienna

From 1751 to 1762 Auenbrugger was connected with the Spanish Hospital at Vienna, first as assistant, later as physician In 1754 he first noted the difference in sounds produced by striking the wall of the chest in various places Not until he had pursued the line of clinical research this opened up to him for seven years did he publish his results and observations The 95-page book, "Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi," was published in Vienna in 1761, the same year that brought forth Morgagni's "De sedibus et causis morborum per anatomen indagatis libri quinque"

After describing the sound produced by striking the chest of a healthy person in different regions, Auenbrugger gave a detailed account of his method of practicing percussion Had he but applied his ear as well as his hand he would have anticipated Laennec But he came near to the discovery of auscultation when, under part three of his Eleventh Observation in "On percussion of the chest," he wrote "If at this time, while the patient is coughing and spitting, the palm of the

[1]He wrote the libretto for Antonio Salieri's *Der Rauchfangkehrer, oder die unentbehrlichen Verrater ihrer Herrschaften aus Eigennutz,* which was presented at Vienna in 1781 This libretto attracted the attention of the Empress Maria Theresa She asked him to write another, but Auenbrugger replied that he had something better to do than to write operas

hand be placed over the site of the vomica, i e, over the place where its existence had been detected by percussion—the noise of fluid within the chest will be sufficiently manifest''

It is of interest to notice, especially, the diseases which he was able to detect merely by means of the preternatural sounds heard through percussion, diseases whose existence he was later able to confirm by studies at necropsy These diseases included ''scirrhus'' of the lungs, vomica, empyema, pleural effusion, dropsy of the pericardium, extravasation of blood into the cavity of the pleura or pericardium, and aneurysm of the heart

Auenbrugger's discovery was ignored by those occupying the high places of medicine of his day Even his own teacher, Baron van Swieten, for whom Auenbrugger had the highest respect, was not impressed by the remarkable new aid to diagnosis The only well-known physicians in good standing who appreciated Auenbrugger's work at that time were Dr Stoll of Vienna, who both used and taught percussion from 1776 to 1784, and Charles G Ludwig of Leipzig The ''Inventum novum'' was translated into French by Rozière of Montpellier in 1770 And although the book went through two French editions it seems to have been practically unknown until the clinician Jean-Nicholas Corvisart, noted teacher[2] and physician to Napoleon Bonaparte, revived the discovery

It is said that Corvisart had never heard of Auenbrugger and his ''Inventum novum'' until he read of it in the works of Stoll From that time on he practiced percussion with perseverance on living subjects as well as cadavers After twenty years of experience he translated Auenbrugger's work and added his own voluminous commentaries to it in 1808, only a year before Auenbrugger's death Corvisart, in his 440-page translation, gave full credit to Auenbrugger as the discoverer of percussion, but Corvisart's renown at home and abroad quickly placed the discovery of Auenbrugger on a high pinnacle In his preface, Corvisart said ''I declare from experience, that this sign of which I treat is one of the greatest importance, not only in detecting disease, but also in curing it, and therefore merits first place after exploration of the pulse and respiration'' (Quoted from Otis)

Notwithstanding the neglect of Auenbrugger's sign during his own lifetime, it cannot be said that he was not appreciated He had a large practice, was noted for his philanthropy, had a genuine devotion to the science and the art of medicine, and had a high regard for the poor as well as the rich

His life at home was happy He had fallen in love with Marianna von Priestersberg when he was a student, and he married her in 1754 They had two children, both daughters, one of whom was a remarkable pianist, and the other of whom was noted for her beauty and wit

As has been mentioned, Auenbrugger had early found favor with the Empress Maria Theresa, and the Emperor Joseph raised him to noble rank in 1784, probably not because of his discovery but in recognition of his skill as a physician and his services to the public

In the latter part of his life Auenbrugger lost the sight of one eye, but the other was so good that he could tell time by the town clock, which was a considerable distance from his window Two years before his own death his beloved wife, Marianna, died (They had celebrated their golden wedding anniversary in 1804) From that time onward Auenbrugger took little interest in life, remained most of the time in his study and enjoyed only the company of his granddaughters His death, the result of a cold, occurred in 1809 He was in his eighty-seventh year

[2]Laennec was one of his pupils

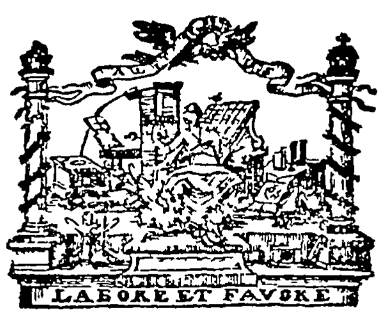
LEOPOLDI AUENBRUGGER

MEDICINÆ DOCTORIS
IN CÆSAREO REGIO NOSOCOMIO NATIONUM
HISPANICO MEDICI ORDINARII.

INVENTUM NOVUM

EX

PERCUSSIONE THORACIS HUMANI
UT SIGNO
ABSTRUSOS INTERNI
PECTORIS MORBOS
DETEGENDI



VINDOBONÆ,

TYPIS JOANNIS THOMÆ TRATTNER, CÆS. REG. MAJEST. AULÆ TYPOGRAPHI.

MDCCLXI.

ON PERCUSSION OF THE CHEST*

By

LEOPOLD AUENBRUGGER

PREFACE

I HERE present the Reader with a new sign which I have discovered for detecting diseases of the chest This consists in the Percussion of the human thorax, whereby, according to the character of the particular sounds thence elicited, an opinion is formed of the internal state of that cavity In making public my discoveries respecting this matter, I have been actuated neither by an itch for writing, nor a fondness for speculation, but by the desire of submitting to my brethren the fruits of seven years' observation and reflexion In doing so, I have not been unconscious of the dangers I must encounter, since it has always been the fate of those who have illustrated or improved the arts and sciences by their discoveries, to be beset by envy, malice, hatred, detraction and calumny

This the common lot, I have chosen to undergo, but with the determination of refusing to every one who is actuated by such motives as these, all explanation of my doctrines What I have written I have proved again and again, by the testimony of my own senses, and amid laborious and tedious exertions—still guarding, on all occasions, against the seductive influence of self-love

And here, lest any one should imagine that this new sign has been thoroughly investigated, even as far as regards the diseases noticed in my Treatise, I think it necessary candidly to confess, that there still remain many defects to be remedied—and which I expect will be remedied—by careful observation and experience Perhaps, also, the same observation and experience may lead to the discovery of other truths, in these or other diseases, of like value in the diagnosis, prognosis and cure of thoracic affections Owing to this acknowledged imperfection, it will be seen, that, in my difficulties, I have had recourse to the Commentaries of the most illustrious Baron Van Swieten, as containing every thing which can be desired by the faithful observer of the nature, by which means I have not only avoided the vice of tedious and prolix writing, but have, at the same time possessed myself of the firmest basis whereon to raise, most securely

*_Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi_, Vienna 1761 Translated by John Forbes M D London, 1824 Reprinted from Sigerist, H E Bull Inst Hist Med 4 379-403 1936

and creditably, the rudiments of my discovery In submitting this to the public, I doubt not that I shall be considered, by all those who can justly appreciate medical science, as having thereby rendered a grateful service to our art, inasmuch as it must be allowed to throw no small degree of light upon the obscurer diseases of the chest, of which a more perfect knowledge has hitherto been much wanted

In drawing up my little work I have omitted many things that were doubtful, and not sufficiently digested, to the due perfection of which it will be my endeavour henceforth to apply myself To conclude, I have not been ambitious of ornament in my mode or style of writing, being contented if I shall be understood

December 31, 1760

FIRST OBSERVATION

OF THE NATURAL SOUND OF THE CHEST, AND ITS CHARACTER IN DIFFERENT PARTS

I

The thorax of a healthy person sounds, when struck

Scholium I deem it unnecessary to give in this place, any description of the thorax I think it sufficient to say, that by this term I mean that cavity bounded above by the neck and clavicles, and below by the diaphragm in the sound state, the viscera it contains are fitted for their respective uses

II

The sound thus elicited (I) from the healthy chest, resembles the stifled sound of a drum covered with a thick woollen cloth or other envelope

III

This sound is perceptible on different parts of the chest in the following manner

1 On the right side anteriorly it is observed from the clavicle to the sixth true rib, laterally, from the axilla to the seventh rib, and posteriorly, from the scapula to the second and third false ribs

2 The left side yields this sound from the clavicle to the fourth true rib, anteriorly, and on the back and laterally, in the same extent as the other side over the space occupied by the heart the sound loses part of its usual clearness, and becomes dull

3 The whole sternum yields as distinct a sound as the sides of the chest, except in the cardiac region where it is somewhat duller

4 The same sound is perceptible over that part of the spinal column which contributes to form the chest

Scholium The sound is more distinct in the lean, and proportionably duller in the robust, in very fat persons it is almost lost The most sonorous region is from the clavicle to the fourth rib anteriorly, lower down, the mammae and pectoral muscles deaden the sound Sometimes, owing to the presence of muscle, the sound is dull beneath the axilla In the scapular regions on the back, owing to the obstacle afforded by the bones and thick muscles there, it is also less distinct Sometimes, but rarely, it exists over the third false rib—owing, I conceive, to a very unwanted length of the thoracic cavity

SECOND OBSERVATION

OF THE METHOD OF PERCUSSION

IV

The thorax ought to be struck, slowly and gently, with the points of the fingers, brought close together and at the same time extended

Scholium Robust and fat subjects require a stronger percussion, such, indeed, as to elicit a degree of sound equal to that produced, by a slight percussion, in a lean subject

V

During percussion the shirt is to be drawn tight over the chest, or the hand of the operator covered with a glove made of unpolished leather

Scholium If the naked chest is struck by the naked hand, the contact of the polished surfaces produces a kind of noise which alters or obscures the natural character of the sound

VI

During the application of percussion the patient is first to go on breathing in the natural manner, and then is to hold the breath after a full inspiration The difference of sound during inspiration, expiration, and the retention of the breath, is important in fixing our diagnosis

VII

While undergoing percussion on the fore parts of the chest, the patient is to hold his head erect, and the shoulders are to be thrown back, in order that the chest may protrude, and the skin and muscles be drawn tight over it a clear sound is thus obtained

VIII

While we are striking the lateral parts of the chest, the patient is to hold his arms across his head, as, thereby, the thoracic parietes are made more tense, and a clearer sound obtained

IX

When operating on the back, you are to cause the patient to bend forwards, and draw his shoulders towards the anterior parts of the chest, so as to render the dorsal region rounded, and for the same reasons, as stated in VIII

Scholium Any healthy person may make experience of percussion in his own person or that of other sound subjects, and will thus be convinced from the variety of the sounds obtained, that this sign is not to be despised in forming a diagnosis

THIRD OBSERVATION

OF THE PRETERNATURAL OR MORBID SOUND OF THE CHEST, AND ITS GENERAL IMPORT

X *and* Scholium

To be able justly to appreciate the value of the various sounds elicited from the chest in cases of disease, it is necessary to have learned by experience on many subjects, the modification of sound, general or partial, produced by the habit of body, natural conformation as to the scapulae, mammae, the heart, the capacity of the thorax, the degree of fleshiness, fatness, etc, etc, inasmuch, as these various circumstances modify the sound very considerably

XI

If, then, a distinct sound, equal on both sides, and commensurate to the degree of percussion, is not obtained from the sonorous regions above mentioned, a morbid condition of some of the parts within the chest is indicated

Scholium On this truth a general rule is founded, and from this certain predictions can be deduced, as will be shown in order For I have learned from much experience that diseases of the worst description may exist within the chest, unmarked by any symptoms, and undiscoverable by any other means than percussion alone

A clear and equal sound elicited from both sides of the chest indicates that the air cells of the lungs are free, and uncompressed either by a solid or liquid body (Exceptions to this rule will be mentioned in their place)

XII *and* XIII

If a sonorous part of the chest, struck with the same intensity, yields a sound duller than natural, disease exists in that part

XIV

If a sonorous region of the chest appears, on percussion, entirely destitute of the natural sound—that is, if it yields only a sound like that of a fleshy limb when struck,—disease exists in that region

Scholium The nature of the indications above pointed out, will be understood by any one who attends to the difference of sound elicited by percussion of the chest, and of the thigh, in his own person

XV

The superficial extent of this unnatural sound (XIV) in a sonorous region, is commensurate with the extent of the morbid affection

XVI

If a place, naturally sonorous, and now sounding only as a piece of flesh when struck, still retains the same sound (on percussion) when the breath is held after a deep inspiration,—we are to conclude that the disease extends deep into the cavity of the chest

XVII

If the same results (XVI) are obtained both before and behind, on points precisely opposite, we are to conclude that the disease occupies the whole diameter of the chest

Scholium These varying results depend on the greater or less diminution of the volume of air usually contained in the thorax (lungs), and the cause which occasions this diminution, whether solid or liquid, produces analogous results to those obtained by striking a cask, for example, in different degrees of emptiness or fulness the diminution of sound being proportioned to the diminution of the volume of air contained in it

FOURTH OBSERVATION

OF THE DISEASES IN GENERAL IN WHICH THE MORBID SOUND OF THE CHEST IS OBSERVED

XVIII

The preternatural or morbid sound occurs in acute and chronic diseases, it always accompanies a copious effusion of fluid in the thoracic cavity

Scholium It must be admitted that whatever diminishes the volume of air within the chest, diminishes the natural sound of that cavity, but we know from the nature, the causes, and the effects, of acute and chronic diseases of the chest, that such a result is possible in these cases, and the fact is finally demonstrated by examinations after death The effect of effused fluids in producing the morbid sound, is at once proved by the injection of water into the thorax of a dead body, in which case it will be found that the sound elicited by percussion, will be obscure over the portion of the cavity occupied by the injected liquid

FIFTH OBSERVATION
OF ACUTE DISEASES IN WHICH THE CHEST YIELDS THE MORBID SOUND

XIX

The morbid sound which is observed in acute diseases, occurs during their progress, or at their termination

Scholium This consideration ought to lead all medical men to use percussion in acute diseases, as they will thereby be enabled to form a more correct judgment, which in such cases is always a matter of difficulty It has often occurred to me to see cases of acute diseases, apparently over, and imposing on the physician under the mask of intermittent or remittent fevers, and which have eventually ended in a fatal vomica or fatal schirrus of the lungs

XX

The preternatural sound which is perceived during the course of acute diseases of the chest, occurs most frequently in inflammatory affections

Scholium The reason of this observation (XX) will be obvious to any one acquainted with the nature of inflammation The preternatural sound may also be observed sometimes in epidemic exanthematous diseases, previously to the eruption,—as was the case in the petechial epidemic of 1757, 1758, 1759, and in the miliary epidemic of the present year (1760) In the latter instance, I observed that the preternatural sound, when once present, continued to the termination of the eruption

XXI

The morbid sound which occurs towards the termination of acute diseases, is observed, when the excretion of morbid matter is not adequate to the severity of the affection

XXII

The morbid sound occurring in inflammatory diseases is commonly observable on the fourth day, it rarely precedes, but often follows this period

Scholium This sign occurs rarely on the third, and very often on the fourth, fifth, and seventh day—but never later It is observed in those inflammatory affections of the pleura or lungs, or both, which are accompanied by a humid cough, but not in those attended by a dry cough,—such (e g) as the dry pleurisy, and inflammation of the mediastinum, pericardium, and heart At least in these latter affections, the sound is not observed, until such time as they verge towards a fatal termination, or have degenerated into obvious abscesses or vomicae

XXIII

The morbid sound increases, from the time of its appearance, according to the nature, severity, and duration of the disease, it diminishes proportionably to the nature, duration, and copiousness of the excretions

Scholium The progressive augmentation of the preternatural sound depends on the gradual deposition of the morbific matter, which I have often found in such quantity as to occupy the inferior two-thirds of the affected side

XXIV

The disease in which the preternatural sound is once present, either proves fatal [on a decretory day, reckoning from its origin], passes off with due excretion, or terminates in other affections

XXV

The following corollaries are the result of my observation of inflammatory diseases of the chest, studied under the sign of morbid resonance

1 The duller the sound, and the more nearly approaching that of a fleshy limb stricken, the more severe is the disease

2 The more extensive the space over which the morbid sound is perceived, the more certain is the danger from the disease

3 The disease is more dangerous on the left than on the right side

4 The existence of the morbid sound on the superior and anterior part of the chest (i e from clavicle to the fourth rib) indicates less danger, than on the inferior parts of the chest

5 The want of the natural sound behind, indicates more danger than it does on the anterior and superior part of the chest

6 The total destitution of sound over one whole side, is generally (passim) a fatal sign

7 The absence of sound along the course of the sternum is a fatal sign

8 The entire absence of the natural sound over a large space in the region of the heart, is a fatal sign

Scholium I have sometimes observed that the fatal prognostics given in the corollaries 6 and 7, were not verified when the matter made its way outwards, or abscesses formed in parts less essential to life And this natural process has been often happily imitaded by the ancients, by cauterising or otherwise incising, the affected parts

SIXTH OBSERVATION

OF CHRONIC DISEASES IN WHICH THE PRETERNATURAL SOUND IS OBSERVED

XXVI

The preternatural sound observed in chronic diseases is owing either to (1) some hidden condition of the organs, which disorders them with a slow progress and finally destroys them, or exists (2) when certain obvious causes have induced a slow disorganisation of the same

Scholium These are the general sources of chronic diseases of the chest, and from whichever of the two classes of causes these arise, the morbid sound will equally and always be present

XXVII

The diseases of the first class are, (1) those which depend on hereditary predisposition, (2) those which arise from affections of the mind, particularly ungratified desires, the principal of which is Nostalgia, (3) those which affect certain artisans, naturally possessing weak lungs

Scholium 1 The influence of an hereditary taint in producing diseases we know by experience, though we cannot explain it —See Van Swieten

2 Mental affections, we find, produce quite opposite effects, while acting as causes of pectoral diseases Of these affections of the mind I have observed none more powerful in rendering obscure the natural resonance of the chest, than the destruction of cherished hopes And as among this class of diseases, Nostalgia (commonly called *heimwehe*—home ail) occupies the first place, I shall here give a short history of it

When young men, not yet arrived at their full growth, are forcibly impressed into the military service, and thereby at once lose all hope of returning safe and sound to their beloved home and country, they become sad, silent, listless, solitary, musing and full of sighs and moans, and finally quite regardless of, and indifferent to, all the cares and duties of life From this state of mental disorder nothing can rouse them,—neither argument, nor promises, nor the dread of punishment, and the body gradually pines and wastes away, under the pressure of ungratified desires, and with the preternatural sound of one side of the chest This is the disease Nostalgia I have examined the bodies of many youths who have fallen victims to it, and have uniformly found the lungs firmly united to the pleura, and the lobes on that side where the obscure sound had existed, callous, indurated, and more or less purulent Some years ago, this disease was very common, but is now rarely met with, since the wise arrangement has been adopted of limiting the period to military service to a certain number of years only

3 The various arts and occupations of life have their peculiar diseases, in like manner as the ages, temperaments, and sexes have theirs This truth is exemplified in the case of the man of letters, the husbandman, the workers in metals, painters, etc, etc Our particular business, however, at present, is with those arts which dispose to diseases of the chest indicated by the sound so often described Thus I have remarked that Tailors, Millers, etc who are forced to inhale, during their labours, a fine dust, become phthisical, while shoemakers, weavers, etc from the forced position or application of their weak chests, during their various occupations, become asthmatical, with scirrhous lungs, etc

I may here state a fact which I have frequently proved by dissection, but which I cannot well account for—it is this in the above mentioned class of cases it is extremely rare to find both lungs affected at the same time, and, when this happens, one lung is always more diseased than the other

XXVIII

The diseases mentioned (in XXVI, 2) arise either from (1) a vitiated condition of the fluids, gradually produced, or (2) from acute affections imperfectly cured

Scholium 1 The vitiation of the humours arises from ingesta which cannot be assimilated, the effect of which in producing chronic diseases is well known

2 An acute disease is said to be imperfectly cured when some morbid affection still remains after it, in some part of the body This morbid condition will be observed either in the site of the primary disease, or, at least, in that portion of the chest, which yields the morbid sound,—namely, the pleura, or lungs, or both these together, or the mediastinum or pericardium When the primary inflammatory disease is succeeded by a collection of pus in the chest, the affection is readily recognized, but if the secondary affection is a scirrhus of the lung, how often and how grievously are medical men thereby deceived! Often have I met with cases of fancied convalescence from acute fevers, in which there was hardly any cough or dyspnoea, or indeed any other sign of disease (as appeared to the attendants) but a trifling degree of irregular fever In these cases, however, on percussion the preternatural sound was found over one whole side of the chest, and the final result was death, preceded either by dropsy or extreme emaciation, the real seat of the disease remaining, perhaps, unknown to the very last!

XXIX

For the above reasons, it may be received as a general rule in chronic diseases, that when, together with the indication stated XXVI, there are emaciation and debility,—the case is desperate

Scholium This result is inevitable whensoever the disease does not yield to medicine In such cases we may always conclude, that the lung of the side which yields the preternatural sound, is either compressed by some foreign body, is indurated by disease, or destroyed by some morbid acrimony developed within its own structure

SEVENTH OBSERVATION

OF THE PRETERNATURAL SOUND OF THE CHEST WHICH RESULTS FROM COPIOUS EXTRAVASATION OF THE FLUIDS CONTAINED IN THE VESSELS OF THAT CAVITY

XXX

The fluids contained in the vessel of the chest are 1 Chyle, 2 Blood, 3 Serum and Lymph

Scholium I must candidly admit that I have never seen a case of extravasated chyle I however believe the thing possible, although I am well aware that the thoracic duct runs outside the pleura the same causes that produce erosion and perforation of the thoracic parietes, may produce this

XXXI

The extravasation of these fluids (XXX) may arise from the following causes (1) rupture of the containing vessels, (2) too great tenuity of the contained fluids, (3) nonabsorption of the same, etc, etc

Scholium 1 Under this head come wounds, contusions, etc

2 Extravasations from internal causes arise from rupture of relaxed and debilitated vessels, during a state of plethora and overactivity of the circulation

3 A third class of causes are obstructions originating in a bad habit of body

XXXII

When from these causes the fluids mentioned are poured out in considerable quantity, the preternatural sound will exist over the space occupied by them

Scholium The correctness of this statement is evinced by the experiment mentioned at the end of the scholium of XVII

According to the plan formerly proposed (XI), I shall now proceed to notice those affections of the chest which are not indicated by percussion

EIGHTH OBSERVATION

OF THOSE AFFECTIONS OF THE CHEST WHICH ARE NOT INDICATED BY PERCUSSION

XXXIII

Certain diseases attended by a violent cough, and thereby creating a suspicion that the lungs are certainly implicated, are nevertheless truly diseases of the abdomen, and affect the pulmonary organs merely sympathetically

Scholium Under this head are ranged the gastric and convulsive coughs of infants, pregnant women, and such other persons as have their abdominal viscera oppressed by the lentor of autumnal agues, or a superfluity of phlegm

XXXIV

Violent coughs, dyspnoeas, asthmas and consumptions, are also occasionally observed which originate in some incomprehensible irritability of the nerves of the chest

Affections of this sort rarely give rise to the preternatural sound from the absence of this, however, and the presence of a copious watery urine, their existence may be pretty confidently presumed

Scholium Under this head are ranged the coughs, dyspnoeas, and asthmas so common in hysterical and hypochondriacal affections, the nervous consumption and asthma of old persons, and, perhaps we may *add the polypous concretions found near the heart in young subjects*

XXXV

A slight engorgement of the lung, a scirrhus of small extent, a small vomica, and a trifling extravasation, are not detected by percussion,—unless, sometimes, by the decreased resonance of the affected part

Scholium These affections are not dangerous until they reach a size when they become more readily discoverable by means of percussion

XXXVI

There is another class of diseases of the lungs [undiscoverable by percussion] in which the distinguishing symptoms are a very severe cough, with expectoration of fatty, chalky, gypseous and stony matters

Scholium The cases are known by the nature of the expectoration I have frequently observed a cough of this kind (but without the peculiar expectoration) succeeding miliary fevers improperly treated

NINTH OBSERVATION

OF THE APPEARANCES ON DISSECTION, IN CASES WHERE THE PRETERNATURAL SOUND OF THE CHEST HAD BEEN OBSERVED

XXXVII

These are the following

1 Scirrhus of the lungs,

2 The conversion of this into an ichorous vomica,

3 A purulent vomica (simple or ruptured) in the pleura, lungs, mediastinum or pericardium

4 Empyema,

5 Dropsy of the chest, in one or both cavities,

6 Dropsy of the pericardium,

7 Extensive extravasation of blood in the cavity of the pleura or pericardium,

8 Aneurism of the heart

Scholium I will now proceed to notice these diseases in order, premising, occasionally, some account of the general symptoms

TENTH OBSERVATION

OF SCIRRHUS OF THE LUNGS, AND ITS SYMPTOMS

XXXVIII

By scirrhus of the lungs I mean the degeneration of the natural spongy substance of the organ into an indolent fleshy mass

Scholium A portion of sound lung swims in water, but this carniform degeneration sinks There is often observed a vast difference in the character of these scirrhi, in respect of hardness, colour, and component parts Thus, in inflammatory diseases of the chest proving fatal on the fifth, seventh, or ninth day, the lung is very often found so completely gorged with blood, as to resemble liver in every respect, both as to colour and consistence One appearance deserves to be noticed the lung is frequently invested with a purulent adventitious membrane, in those instances wherein the fatal peripneumony has succeeded an acute pleurisy In chronic diseases of the lungs the appearances are extremely various Frequently they are interspersed and as it were marbled with a fatty kind of matter, frequently along with the fleshy appearance, they have the consistence of cartilage, and very often they are found indurated by means of a thickened and black blood These varieties, doubtless, depend on varieties of the morbific matter

XXXIX

The presence of scirrhus of the lungs, in its primary unsoftened condition, may be suspected from the following signs

Together with the diminution or entire loss of the natural sound over the affected part, there is an infrequent cough without any expectoration, or with only a scanty excretion of viscid and crude sputa During a state of quiescence there is nothing to be observed much amiss, either in the condition of the pulse or respiration, but upon any considerable bodily motion, or after speaking for some time, these persons become speedily exhausted, anxious, and breathless, and complain of a sense of dryness and roughness in the throat At the same time the pulse, which had previously been of moderate frequency, becomes quick and unequal, the respiration and speech are broken and interrupted by sighs, the temporal, sublingual, and jugular veins of the affected side, are more than usually distended, while it will be observed that this side of the chest is less moveable than

the other, during inspiration Meanwhile the natural and animal functions continue to be well performed, and the patient can lie on either side indifferently

All the above symptoms are more severe in proportion as the scirrhus is more extensive

ELEVENTH OBSERVATION

OF VOMICAE IN GENERAL

XL

When an humour, sound or morbid, is deposited from the circulating mass in a solid form, and (together with the extreme vessels) is afterwards, by means of the vital powers softened and converted into matter, and contained in a sort of capsule, I term this collection of matter a Vomica

Scholium This notion applies to every vomica, whether produced by a vice of the solids or fluids, as is clear from the history of obstruction and inflammation

XLI

I have observed two kinds of Vomica—the Ichorous and Purulent The former occupies the lungs only, the latter, both the lungs and other thoracic viscera They are both either close, or communicating with the Trachaea

Scholium By the term Ichorous Vomica, I mean a sac containing a thin fluid frequently of a reddish yellow colour, frequently of a reddish brown, often of a colour between these, different from pus, and arising from the destruction of a scirrhus lung By Purulent Vomica, I understand an encysted abscess of the chest, resulting from the conversion of an inflamed spot into a white, thick, glutinous, fatty matter When these communicate with the Bronchia and discharge any of their contents by expectoration, they are called open, otherwise, close or shut

XLII

1 Ichorous Vomica —If a scirrhus of the lung, recognized by its proper signs (XXXIX) is converted into matter, it presents the following symptoms The patient begins to languish and waste away insensibly (although the usual quantity of food is taken), with a quick, contracted, and unequal pulse The respiration, even during a state of quietude, is unnaturally anxious and frequent, and is remarkably interrupted by sighing The forehead, during the more severe attacks, is sometimes covered with a cold sweat The eyes are dim, the veins of the cheeks and lips are livid, and the tongue, especially on the affected side, is of a leaden hue At the same time there is neither pain nor thirst The diseased side, however, is observed to be less mobile than natural, and the degree of immobility is proportioned to the bulk of the vomica into which the scirrhus has been

resolved The cough is infrequent, interrupted, and dry, or the expectoration, if any, is dirty or blackish (coenosum aut fuscum)

When things have got to this height, the appetite begins to fail, and at length is entirely lost, and whatever is eaten only produces an increase of anxiety during the process of digestion this process however, takes place without any hectic flushing, which always accompanies the purulent vomica

In some cases, when there is a dissolution of the central parts of the scirrhus, the abdomen and hypochondries sink in, in a very few instances, the same parts are slightly swollen, and with an indistinct feeling of fluctuation The urine rarely presents any deviation from the natural state, sometimes, however, it is red, and with a sediment (if any exists) of a cinnabar colour The stools are of natural character, except under the influence of medicine The extremities, even when of a livid colour, are never hotter than natural, until a few days before death, the affected side is, moreover, observed to swell, and the hand and foot in the first place The patient now suffers from frequent sinkings and faintings, and from having hitherto been able to lie easily on either side, he is able to remain on the affected side only

2 Close Purulent Vomica—The following are the symptoms of this affection While the abdominal organs still continue to perform their functions well, there is often present a very troublesome, frequent, dry cough, so severe as to irritate the fauces, to render the voice hoarse, and often to excite vomiting At this time are observed frequent irregular chills, followed by heat, and strong flushing of the cheeks and lips, particularly of the affected side A degree of lassitude is experienced, more remarkable after a full meal, and at the same time there is perceived a degree of quickness and straitness of the respiration, sufficient to excite suspicion of some morbid affection of the chest The pulse is also found to be contracted, frequent somewhat hard and unequal during the period of digestion, and even at other times it is never in a perfectly natural state,—more especially under the influence of bodily motions, laughing, or speaking

If at this time the Vomica has reached a size to be detected by percussion, the following additional signs exist The patient is not nourished by the food taken, partly because it is, in a greater or less degree, rejected by vomiting, and partly on account of the imperfect assimilation of what is retained As the disease increases, the whole process of respiration is at length carried on by one lung, an incessant state of anxiety prevails, and the patient remains fixed on the diseased side, through dread of impending suffocation if he turn on the other The face, hands, feet, and the affected side are oedematous, while the opposite side of the body, from deficient assimilation, hectic heat, and nocturnal perspirations, is extenuated The urine now becomes scanty, red turbid, with a copious branny sediment and soon putrefies, and the

scene is finally closed, with short and asthmatic breathlessness, lividity of the cheeks, lips, and nails, etc

3 Purulent Vomica communicating with the Trachea —When a Vomica of considerable size, discoverable by percussion, bursts into the Trachea, or rather Bronchia, by a large opening, it produces instant suffocation, if by a small aperture, it is recognized by the following marks By means of a violent cough, pus is expectorated, which is, in different cases, white, yellow, saffron, green, brown, bloody, which sinks in water, and, when thrown on hot coals, emits a stinking nidorous smell If at this time, while the patient is coughing and spitting, the palm of the hand be placed over the site of the vomica, i e over the place where its existence had been detected by percussion,—the noise of fluid within the chest will be sufficiently manifest This kind of expectoration will cease for some days, with relief to the patient, but it speedily returns, and is always preceded, for four and twenty hours, by an increase of the febrile state During this state of things, and before the return of the expectoration, if percussion is applied over the site of the vomica a sound exactly like that from a fleshy limb is obtained, but if this is delayed until the evacuation of the accumulated pus, then there is perceived a distinct, though obtuse sound The slow fever which invariably accompanies this condition, is encreased after eating, and is still higher during the night, and at these times, the forehead, neck, and chest, are covered with perspiration With the increase of these symptoms, and the continuation of the purulent expectoration, the breath becomes tainted, insomuch as to be extremely disagreeable both to the patient and the attendants The thirst continues great, but the appetite is lost, even for the greatest delicacies, which, however sparingly taken, produce, in place of refreshment, langour and anxiety (The case is very different with them whose sputa are inodorous, the appetite in many being even great) The urine is *uniformly frothy*, grows speedily putrid, and deposits a viscid, tenacious, white sediment The patient now daily grows more emaciated the bones almost pierce the skin, the hair falls off, the nails become curved, the legs swell, at length a colliquative diarrhaea supervening first lessens, and then suppresses the expectoration, and the sufferer finally dies suddenly, on the third day after that on which he began to remain obstinately fixed on his back, with his legs drawn under him

XLIII

Empyema —When a vomica (XXXVII), ascertained by percussion, discharges its contents into the cavity of the pleura, and upon the diaphragm, Empyema is produced

Scholium I premise this definition to prevent the affection now in question from being confounded with a vomica that has discharged its contents into the trachaea

XLIV

If a large vomica, whose superficial and central extent is supposed to have been recognized by the marks pointed out (Obs Third XV, XVI, XVII), shall have burst as above mentioned (XLIII), it may be recognized by the following signs

The patient who had usually lain on the affected side, starts up with a sudden pain (as if nearly suffocated), and begs to be held in the erect posture

If percussion is now applied, it will be found that the natural sound, which had been nearly lost in the site of the vomica, has in some degree been restored in that place, while it is more or less destroyed (according to the quantity of pus effused) over the posterior and inferior parts of the chest

There is now a very frequent cough, which is either dry, or with a scanty, frothy and noisy expectoration The respiration becomes very laborious, with frequent faintings, and a cold sweat bedews the forehead and throat, the cheeks and lips are of an ominous red, while the nails grow livid, the pupils dilate, and death (which follows in a few hours the rupture of a large vomica) is finally preceded by dimness of sight, etc

A small vomica, ruptured in the same manner, produces the same symptoms, and is equally fatal This issue, however, is of later occurrence, and is preceded by the marks of pleuro-peripneumony

TWELFTH OBSERVATION

OF DROPSY OF THE CHEST

XLV

When water is collected in the cavity of the chest, between the pleura (costalis) and the lungs, the disease is called dropsy of the chest, and this is said to be of two kinds, namely, according as the fluid occupies one, or both sides

Scholium This is ascertained by percussion in the living subject, and is demonstrated by anatomical examination after death The general symptoms of this disease are chiefly the following

1 Difficult and laborious respiration,

2 A cough at intervals, which is dry, or only attended by sputa of a thin watery nature, or occasionally somewhat viscid,

3 A pulse contracted, somewhat hard, frequent, unequal and often intermitting,

4 A sense of breathlessness and suffocation on the slightest motion,

5 An incipient dislike of warm food,

6 Perpetual anxiety about the scrobiculus cordis

7 Great pressure on the chest, and distention of the stomach during the period of digestion,

8 A murmuring noise about the hypochondries, and frequent eructation of flatus, with momentary relief,

9 Scarcely any thirst,

10 Urine very scanty, and rarely made, red, with a lateritious sediment,

11 Swelling of the abdomen, more especially in the Epigastrium, and particularly in that point on which the incumbent water gravitates,

12 A sublivid swelling of the extremities, especially of the feet, which are moreover cold to touch,

13 Oedematous tumescence of the inferior palpebrae,

14 A pallid, or, according to the nature of the affection, a sublivid discoloration of the cheeks, lips, and tongue,

15 Inability to lie down, anxious distressing nights, with heaviness, yet frequently sleepless

All these symptoms vary in a wonderful manner according to the disease

First Kind —Dropsy of one side of the Chest Beside the general signs of this disease above enumerated, the affected side, if completely filled with water, is enfeebled (effoeminatum), and appears less moveable during inspiration In this case, also, the affected side yields no where the natural sound on percussion If the chest is only half-filled, a louder sound will be obtained, over the parts to which the fluid does not extend, and, in this case, the resonance will be found to vary according to the position of the patient, and the consequent level which the liquid attains The Hypochondre of the affected side is also unusually tumid, and more resisting to pressure than the rest of the abdomen The palpebra, hand, and foot of the affected side are slightly oedematous It is a remarkable fact, that the reclining posture (decubitus declivis) is easily borne when the chest is entirely full, while the contrary is the case, when there remains space for the fluctuation of the water

Second Kind —Dropsy of both sides of the Chest If fluid is contained in both sides of the chest, the following specific signs, in addition to the general symptoms, exist The natural sound is destroyed over the space occupied by the water in either side The patients uniformly become asthmatic, and resemble, in many respects, those labouring under Ascites, only that the former have their inferior palpebrae and hands swollen They cannot lie in an horizontal posture, and are equally threatened with suffocation on whichever side they turn, on which account, they are forced to remain sitting, day and night, to prevent the pressure of the fluid from being felt on the upper parts of the chest (which would be the case on lying down), in the same degree on which it now gravitates on the abdomen The effect of this state of things might lead to the sus-

picion of Ascites, only that we find, on examining the patient in the erect position, that the hypochondriac regions are more swollen than the inferior parts, which is not the case in Ascites

All these subjects die as if from peripneumony, that is to say,—the pulse fails, the whole body, except the chest and head, grows cold, the cheeks and extremities become livid, the respiration is at first laborious, then interrupted, and finally ceases altogether

XLVI

Dropsy of the Pericardium—When the liquor pericardii is morbidly increased, so as to be capable of disturbing the natural action of the heart, the disease is called Dropsy of the Pericardium of this there are two species, as the fluid is purulent or serous

Scholium The fluid naturally present in the pericardium accumulates in still greater quantity in those who suffer a long protracted mortal agony, as we find on examination after their death But it is not to this accumulation, originating in the relaxation of death, but to that produced by obstruction during life, that I apply the term dropsy I have ventured to divide the affection into two species, because I have often witnessed both of them In the first variety, the heart is rough, and as it were shagged, with a coating of the purulent matter, while in the latter, the organ is only of a paler colour than natural Many may be of the opinion that the purulent dropsy would be better classed under the head of Empyema, but I shall never quarrel about words, when there are appearances to instruct us

Signs of Hydropericardium—Almost all the symptoms which have already been enumerated as accompanying dropsy of the chest generally, accompany this species also in addition to these, however, I have observed the following specific signs of the dropsy of the pericardium —

The sound in the cardiac region, which I have already stated (III 2, 3) to be naturally more obscure than in the other parts of the chest, is now as completely deadened as if the percussion were applied to a fleshy limb A swelling is perceived in the praecordia, which can readily be distinguished, by its superior resistance, from the stomach distended by flatus

The patients fall asleep, while sitting, the body being inclined forwards, but they soon are roused by the unconscious dropping of the head On this account, they complain to all around them of the distressing propensity to sleep which they experience At the same time they suffer from faintings (accompanied by a pulse frequently unequal in respect both of its rythm and volume), and, indeed continue to undergo to the end of their wretched life, and in every position of body, the greatest distress A few days before death, in many cases the neck is swollen and the eyes

become extremely red, as if from crying This state of things is sometimes terminated suddenly by a stroke of apoplexy, or more slowly by leipothymia

The same signs are furnished by percussion in the purulent, as in the proper dropsy of the pericardium, but in the former, the other symptoms are precisely the same as those which exist in the close purulent vomica In the purulent dropsy, the fluid commonly resembles turbid whey,—the thicker portions of it (quod purulentum est) being found adhering to the heart like fringes

THIRTEENTH OBSERVATION

OF THE SYMPTOMS OF A COPIOUS EXTRAVASATION OF BLOOD

XLVII

The causes of a large extravasation of blood into the cavity of the chest have been noticed in the Scholium of XXXI The following are the symptoms of this affection

Scholium There is incessant and indescribable anxiety and oppression at the praecordia and on the chest, while there is constant jactitation of the body, and complete intolerance of the horizontal posture Percussion elicits none of the natural sound over the space occupied by the extravasated blood In all cases the pulse is extremely contracted, frequent, and irregular in every way The respiration is extremely laborious, with a frequent cough, and broken by profound sighing All the veins become flaccid, and the eyes are at first red but ultimately pale Cold sweats etc follow, and the patient dies stertorous

These are the symptoms when the blood flows into the cavity of the pleura without any accompanying lesion of the lungs when these are wounded, there is also bloody expectoration, and a passage of air to and from the wound in the parietes of the chest

FOURTEENTH OBSERVATION

OF ANEURISM OF THE HEART

XLVIII

When the heart becomes so much distended by blood, accumulated in its auricles and ventricles, as to be unequal to propel forward its contents, it frequently becomes thereby enormously dilated This dilatation has been called Aneurism of the Heart

Scholium We frequently observe this state of the heart on dissection, (1) in sudden and extensive peripneumonies of both lobes at the same time, and (2) in those fatal inflammatory diseases which are noticed towards the end of the Scholium on XXII

The pathognomonic sign of this affection is the complete fleshy sound on percussion existing over a considerable space in the region of the heart Whenever this sound is perceptible in the acute peripneumony it is a sign that the patient will not survive twenty-four hours in fact, he is already at the last gasp, and is speedily carried off as in apoplexy, unconscious of his fate

In the second class of inflammations, the sign is equally fatal, but is attended by different symptoms In this case, the patients suffer dreadfull anxiety and by the constant jactitation of their limbs, are perpetually uncovering themselves Older persons, indeed bear more tranquilly their sufferings, but the younger are pertinaciously restless and violent, struggling and talking, attempting to get out of bed, demanding their clothes, and endeavouring to walk or go about their usual occupations Meanwhile the eyes become dull, the cheeks livid, and the nails and extremities are tinged with a leaden hue, and death is ushered in by cold sweats, and the gradual extinction of the pulse and respiration

Cedant haec miseris in solatium, veris autem medicinae cultoribus in incrementum artis Quod opto!

1772

WILLIAM HEBERDEN

ON ANGINA PECTORIS

WILLIAM HEBERDEN

Portrait by Sir William Beechey, R A , in the Royal College of Physicians, London

(Courtesy Charles C Thomas)

WILLIAM HEBERDEN

(1710-1801)

"Good readers are almost as rare as good authors"

—William Heberden

DURING the Eighteenth Century, which was characterized in medical history as an age of theories and individual systems, few medical men were outstanding Except for the original contributions of such men as Morgagni, Hales, John and William Hunter, Kaspar Friedrich Wolff, and Jenner, as Garrison has shown (p 310), the century represented a period of retrogression Too few physicians were practicing bedside medicine, and the ideas which Harvey, Bacon, and Newton had put forth during the preceding century had not fired the general practitioners with enthusiasm One of the reasons, other than his singular accomplishments, why William Heberden is given a conspicuous place in medical history is that he made it a point to battle the dogma and tyrannic ideologies of many of his contemporaries who were still wrangling over the medical interpretations of the ancient writers Moreover, in his medical practice, which was very extensive, Heberden had the modern attitude of first studying the patient as an individual and later relegating his illness or disease to its proper place in the classification of disease

William Heberden was born in London He was next to the youngest son of Richard and Elizabeth Cooper Heberden His preliminary education was obtained at the Grammar School of St Saviour, Southwark He entered St John's College, Cambridge, in 1724 In 1728 he was granted the degree of Bachelor of Arts Two years later he obtained a fellowship in the same college In 1732 he received the degree of Master of Arts From that year on, he seems to have directed his attention to medical studies, and he received his degree of Doctor of Medicine in 1739

During his stay in medical school and while he was studying materia medica, Heberden was engaged in writing "An Essay on Mithridatium and Theriaca" In that work he exposed the use of mithridatics as antidotes for poisons, so that these glamorous drugs thereby lost their importance The work was not published until 1745 It is of interest in the present account to note Heberden's broad acquaintance with the classics Not only did he call on Hippocrates to elucidate some points, but he also quoted from Homer, Plautus, Virgil, Juvenal, and others

In 1746 Heberden was elected a fellow of the Royal College of Physicians, and in 1748 he began the practice of medicine in London Heberden was further honored in 1749 when he was elected a fellow of the Royal Society Some of his most valuable contributions to medicine subsequently were published in the "Philosophical Transactions" of the Royal Society

In 1752 Heberden married Elizabeth Martin, daughter of John Martin, a prominent citizen and a member of Parliament His wife died in 1754, leaving him with two sons John, who died in infancy, and Thomas, father of the well-known physician, Thomas Heberden In 1760 William Heberden married again, this time to Mary Wollaston By his second marriage he had eight children, only two of whom survived their father These were Mary and William, the latter of whom later became physician to the King

Heberden, in his practice of medicine, stressed the importance of experience in the study and treatment of disease. He makes this interesting comment, "... the practice of physic has been more improved by the casual experiments of illiterate nations, and the rash ones of vagabond quacks, than by the reasonings of all the once celebrated professors of it, and theoretic teachers in the several schools of Europe: very few of whom have furnished us with one new medicine, or have taught us better to use our old ones, or have in any one instance at all improved the art of curing diseases."[1]

From all published accounts it seems that both Heberden's professional and social contacts were pleasant. He treated William Cowper and Bishop Warburton, but perhaps his most famous patient was Samuel Johnson, whom he attended in his final illness. Johnson, in the codicil to his will, left Heberden one of his books. According to a note by Nichols in Boswell's "Life of Samuel Johnson,"[2] Dr. Johnson once called Heberden, "Dr. Heberden, ultimum Romanorum, the last of our learned physicians." Among Heberden's friends and colleagues were John and William Hunter, Fothergill, Jenner, Sir George Baker, Withering, Pitcairn, and Robert Gooch. He also was acquainted with Benjamin Franklin, who induced him to publish for the American colonies a most interesting pamphlet,[3] giving instructions for inoculation for the prevention of the smallpox (1759). According to Pettigrew, Heberden was also on intimate terms with the chief literary men of his day, among them Gray, Jacob Bryant, Mason, Cavendish, Bishop Hurd, Bishop Lowth, Dr. Kennicott, Dr. Jortin, Tyrwhitt, and Stuart. Heberden himself translated the plays of Euripides.

George III thought well of Heberden and, upon Queen Charlotte's arrival in England in 1761, he was named physician to her, an honor which he declined because he felt it might interfere with life as he wished to live it.

In 1778 Heberden was elected an honorary member of the Royal Society of Medicine of Paris. He retired from the practice of medicine in 1782. His last paper, "Of the Measles," was read at the College of Physicians in 1785. In 1787, being still rather active, he was elected vice-president of the Royal Humane Society. He had retired years ago, of course, from active practice and, as he declares in his preface to the "Commentaries," a preface which because of its beautiful sentiment and sage advice we have chosen to reprint in this book, he spent his last years in teaching what he knew to his sons. Dr. Heberden died in Pall Mall on May 17, 1801, in his ninety-first year. Included in LeRoy Crummer's prefatory essay to the publication of the Heberden manuscript, "An Introduction to the Study of Physic," is a list of Heberden's published writings as well as a check list of Heberden's manuscripts.

Heberden's major opus was, of course, his "Commentaries on the History and Cure of Diseases," published posthumously by his son, William. The Royal College of Physicians owns the original two volumes of manuscript which Heberden had carefully written in Latin. According to Crummer (p. 11-12), each volume contains about the same material. William Heberden, Jr., in 1802 chose to have published one of these volumes, which had been completed by the senior Heberden in 1782. The younger Heberden also translated the English edition of the "Commentaries," published the same year (1802). There were many subsequent editions of the work in both languages, for it was one of the most popular books on medicine of the first part of the Nineteenth Century. For those who are interested in the evolution of medical

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[1]Pettigrew, Thomas J.: 'William Heberden,' in *Medical portrait gallery*, London, 1840, Whittaker and Co., vol. 3, pp. 11-12.

[2]Boswell, James: *The Life of Samuel Johnson*, Bath, England, George Bayntun, 1925, vol. 2, p. 1013.

[3]Heberden, William: *Plain instructions for inoculation in the small-pox, by which any person may be enabled to perform the operation, and conduct the patient through the distemper*. Printed at the expense of the author, to be given away in America. London, 1759, 12 pp.

thought, this book holds many fascinations In Heberden's description of disease the keen, trained logical power of observation not surpassed by Hippocrates or Sydenham is noticed The book represents a careful analysis of the professional experience of more than forty years of active practice of a physician possessed of a high mental endowment In it are contained his original portrayals of varicella which he originally published in 1768,[4] of angina pectoris, a condition which he named and which was first described by him in 1772[5] which we are reproducing, and his notation of the nodules in the fingers which occur in arthritis deformans An actual case of angina pectoris was described in the memoirs of the Earl of Clarendon (1632)[6] and Morgagni[7] presented a description of this disease (1707) It is also of interest to note that at the same time Heberden reported his cases, Dr Rougnon[8] of Besançon wrote a letter about it concerning a captain of cavalry who had d unexpectedly during an attack of pain situated in the retrosternal region The pain appeared after effort, similar attacks having occurred before Several other accounts of angina pectoris were then reported, including Home's account of John Hunter's angina pectoris, which we are also reproducing But none was so clear and concise as the classic of Heberden, who not only gave the disease its name but also gave it a masterful description

[4] Heberden William On the Chicken-Pox Med Tr Roy Coll Phys, London 1 427-436 1768

[5] Heberden William Some account of a disorder of the breast Med Tr Roy Coll Phys London 2 59-67 1772

[6] Case of Henry Hyde 1632 In his *Life* Oxford 1759, p 9

[7] Morgagni J B *De sedibus et causis morborum per anatomen indagatis libri quinque Dissectiones et animadversiones, nunc primum editas complectuntur propemodum innumeras medicis chirurgis anatomicis profuturas* vol 1 p 282 Venice 1761

[8] Rougnon de Magny N F *Lettre a M Lorry touchant les causes de la mort de feu Monsieur Charles ancien capitaine de cavalerie arrivee a Besancon le 2? fevrier* 1768 55 pp

COMMENTARIES

ON

THE HISTORY AND CURE

OF

DISEASES

BY

WILLIAM HEBERDEN, M D

Γέρων, καὶ κάμνειν οὐκέτι δυνάμενος, τοῦτο τὸ βιβλίον ἔγραψα, συντάξας τὰς μετὰ πολλῆς τριβῆς ἐν ταῖς τῶν ἀνθρώπων νόσοις καταληφθείσας μοι πείρας.

ALEX TRALL. Lib XII

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1802

COMMENTARIES ON THE HISTORY AND CURE OF DISEASES*

PREFACE

PLUTARCH says,† that the life of a vestal virgin was divided into three portions, in the first of which she learned the duties of her profession, in the second she practised them, and in the third she taught them to others This is no bad model for the life of a physician and as I have now passed through the two first of these times, I am willing to employ the remainder of my days in teaching what I know to any of my sons who may choose the profession of physic, and to him I desire that these papers may be given

The notes from which the following observations were collected, were taken in the chambers of the sick themselves, or from their attendants, where several things might occasion the omission of some material circumstances These notes were read over every month, and such facts, as tended to throw any light upon the history of a distemper, or the effects of a remedy, were entered under the title of the distemper in another book, from which were extracted all the particulars here given relating to the nature and cure of diseases It appeared more advisable to give such facts only, as were justified by the original papers, however imperfect, than either to supply their defects from memory, except in a very few instances, or than to borrow any thing from other writers

The collections from the notes, as well as the notes themselves, were written in Latin, the distempers being ranged alphabetically, and this is the reason that the titles are here in that language In making the extracts it was not only more easy to follow the order in which the observations had been ranged, but there was likewise less danger of any confusion or omission, and little or no inconvenience can arise from preserving the Latin names of the distempers

An useful addition might have been made to these papers by comparing them with the current doctrine of diseases and remedies, as also with what is laid down in practical writers, and with the accounts of those who treat of the dissections of morbid bodies, but at my advanced age it would be to no purpose to think of such an undertaking

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*Heberden, William *Commentaries on the History and Cure of Diseases*, London, 1802

†Plutarch in Numa. et.

CHAPTER 70

PECTORIS DOLOR*

Beside the asthma, hysteric oppressions, the acute darting pains in pleurisies, and the chronical ones in consumptions, the breast is often the seat of pains, which are distressing, sometimes even from their vehemence, oftener from their duration, as they have continued to tease the patient for six, for eight, for nine, and for fourteen years. There have been several examples of their returning periodically every night, or alternately with a head-ach. They have been called gouty, and rheumatic, and spasmodic. There has appeared no reason to judge that they proceed from any cause of much importance to health (being attended with no fever), or that they lead to any dangerous consequences, and if the patient were not uneasy with what he feels, he needs never to be so on account of any thing which he has to fear.

If these pains should return at night, and disturb the sleep, small doses of opium have been found serviceable, and may be used alone, or joined with an opening medicine, with a preparation of antimony, or with the fetid gums. Externally, a small perpetual blister applied to the breast has been successful, and so has an issue made in the thigh. A large cumin plaster has been worn over the seat of the pain with advantage. The volatile, or saponaceous liniment, may be rubbed in over the part affected. Bathing in the sea, or in any cold water, may be used at the same time.

But there is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned more at length. The seat of it, and sense of strangling, and anxiety with which it is attended, may make it not improperly be called angina pectoris.

They who are afflicted with it, are seized while they are walking, (more especially if it be up hill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or to continue, but the moment they stand still, all this uneasiness vanishes.

In all other respects, the patients are, at the beginning of this disorder, perfectly well, and in particular have no shortness of breath, from which it is totally different. The pain is sometimes situated in the upper part, sometimes in the middle, sometimes at the bottom of the os sterni, and often more inclined to the left than to the right side. It likewise very frequently extends from the breast to the middle of the left arm. The pulse is, at least sometimes, not disturbed by this pain, as I have had opportunities of observing by feeling the pulse during the paroxysm. Males are most liable to this disease, especially such as have past their fiftieth year.

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*Heberden's original publication appeared in 1772. We are reprinting from the *Commentaries* published in 1802.—F. A. W. 1940

After it has continued a year or more, it will not cease so instantaneously upon standing still, and it will come on not only when the persons are walking, but when they are lying down, especially if they lie on the left side, and oblige them to rise up out of their beds In some inveterate cases it has been brought on by the motion of a horse, or a carriage, and even by swallowing, coughing, going to stool, or speaking, or any disturbance of mind

Such is the most usual appearance of this disease, but some varieties may be met with Some have been seized while they were standing still, or sitting, also upon first waking out of sleep and the pain sometimes reaches to the right arm, as well as to the left, and even down to the hands, but this is uncommon in a very few instances the arm has at the same time been numbed and swelled In one or two persons, the pain has lasted some hours, or even days, but this has happened when the complaint has been of long standing, and thoroughly rooted in the constitution once only the very first attack continued the whole night

I have seen nearly a hundred people under this disorder, of which number there have been three women, and one boy twelve years old All the rest were men near, or past the fiftieth year of their age

Persons who have persevered in walking till the pain has returned four or five times, have then sometimes vomited

A man in the sixtieth year of his life began to feel, while he was walking, an uneasy sensation in his left arm He never perceived it while he was travelling in a carriage After it continued ten years, it would come upon him two or three times a week at night, while he was in bed, and then he was obliged to sit up for an hour or two before it would abate so much as to suffer him to lie down In all other respects he was very healthy, and had always been a remarkably strong man The breast was never affected This disorder, its seat excepted, perfectly resembled the angina pectoris, gradually increasing in the same manner, and being both excited and relieved by all the same causes He died suddenly without a groan at the age of seventy-five

The termination of the angina pectoris is remarkable For if no accident intervene, but the disease go on to its height, the patients all suddenly fall down, and perish almost immediately Of which indeed their frequent faintness, and sensations as if all the powers of life were failing, afford no obscure intimation

The angina pectoris as far as I have been able to investigate, belongs to the class of spasmodic not of inflammatory complaints For

In the 1st place the access and the recess of the fit is sudden

2dly, There are long intervals of perfect health

3dly, Wine, and spirituous liquors and opium afford considerable relief

4thly It is increased by disturbance of the mind

5thly It continues many years without any other injury to the health

6thly, In the beginning it is not brought on by riding on horseback, or in a carriage, as is usual in diseases arising from scirrhus, or inflammation.

7thly, During the fit the pulse is not quickened.

Lastly, Its attacks are often after the first sleep; which is a circumstance common to many spasmodic disorders.

Yet it is not to be denied that I have met with one or two patients, who have told me they now and then spit up matter and blood, and that it seemed to them to come from the seat of the disease. In another, who fell down dead without any notice, there immediately arose such an offensive smell, as made all who were present judge that some foul abscess had just been broken.

On opening the body of one, who died suddenly of this disease, a very skilful anatomist could discover no fault in the heart, in the valves, in the arteries, or neighbouring veins, excepting small rudiments of ossification in the aorta. The brain was likewise every where sound. In this person, as it has happened to others who have died by the same disease, the blood continued fluid two or three days after death, not dividing itself into crassamentum and serum, but thick, like cream. Hence when a vein has been opened a little before death, or perhaps soon after, the blood has continued to ooze out as long as the body remained unburied.

With respect to the treatment of this complaint, I have little or nothing to advance: nor indeed is it to be expected we should have made much progress in the cure of a disease, which has hitherto hardly had a place, or a name in medical books.* Quiet, and warmth, and spiritous liquors help to restore patients who are nearly exhausted, and to dispel the effects of a fit when it does not soon go off. Opium taken at bed-time will prevent the attacks at night. I know one who set himself a task of sawing wood for half an hour every day, and was nearly cured. In one also, the disorder ceased of itself. Bleeding, vomiting and purging, appear to me to be improper.

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*Cœlius Aurelianus, as far as I know, is the only ancient writer who has noticed this *complaint, and he but slightly.* "Erasistratus memorat paralyseos genus et *paradoxon* appellat, quo ambulantes repente sistuntur, ut ambulare non possint, et tum rursum ambulare sinuntur."—*Chron.* lib. ii. c. 1.—M. Saussure in his *Voyage dans les Alpes* says, that at the height of 13 or 1,400 toises above the sea, a peculiar tiredness often comes upon those who are ascending such high hills, so that it is impossible to proceed four steps further; and if it were attempted, such strong universal palpitations would come on, as could not fail to end in swooning. Upon resting three minutes, even without sitting down, this tiredness passes, and the power of going on is perfectly restored. The climbing of steep hills, which are not so high above the sea, does not occasion this peculiar fatigue.—Vol. i. p. 482.

1785

WILLIAM WITHERING

AN ACCOUNT OF THE FOXGLOVE

WILLIAM WITHERING

Painting by Carl Fredrik von Breda in the National Museum, Stockholm

(Courtesy Dr Drew Luten)

WILLIAM WITHERING

(1741-1799)

"The Flower of Physicians"

ALTHOUGH he was only fifty-eight years of age at the time of his death, William Withering lived an abundant life, and the period in which he lived was a most interesting one He saw the development of the steam engine, the use of gas lighting in Birmingham, the invention of the spinning jenny and the cotton gin It was his destiny to live through both the American and French Revolutions and to have been sympathetic with the citizen classes who fought for their freedom Moreover, he was a contemporary of such men as the elder Pitt, Burke, Samuel Johnson, Goldsmith, Robert Burns, Voltaire, Washington, Haydn, Beethoven, Mozart, Franklin, and Linnaeus

William Withering was born at Wellington in Shropshire in 1741 According to Cushny, Withering's father, Edmund Withering, was an apothecary, but it is apparent that the father practiced medicine[1] Withering's maternal uncle was a physician in Lichfield His mother's father, Dr George Hector, had delivered Samuel Johnson Thus, early in life, William Withering made contact with the profession which was his eventual choice

Withering was tutored privately at home by the Rev Henry Wood of Ercall Such a method of preliminary education was a common practice in the eighteenth century In 1762 he matriculated at the University of Edinburgh where he studied medicine Edinburgh was the residence of David Hume (1711-1776), the famous Scotch philosopher and political economist, who during Withering's stay in this city was at the height of his brilliant career Among Withering's professors were Hope in botany, Whytt (an authority on hysteria) in medicine, Alexander Monro "Secundus" (who in 1769 discovered the foramen of Monro) in anatomy, and Cullen in chemistry and medicine Withering seems to have been on intimate terms with the last-named professor William Cullen (1712-1790) held the chairs of medicine and chemistry at both Glasgow and Edinburgh He was one of the first to give clinical lectures in Great Britain, and these lectures established a precedent in that they were delivered in English instead of in Latin Cullen was a source of great admiration and inspiration to his pupils and it is of interest to know that he supported Withering in his therapeutic use of foxglove Cullen was the teacher of Benjamin Rush (1745-1813), and it was Rush who introduced into America the complicated system of "fevers" as the basis for most diseases which Cullen had formulated and preached at Edinburgh According to Shryock, the influence of Cullen, as transmitted through the teachings of his pupil, Rush, had an effect on American clinical medicine that endured for a generation Withering received the degree of Doctor of Physic in 1766, his thesis was "De Angina Gangraenosa" (malignant putrid sore throat)

From 1767 until 1775 Withering practiced medicine at Stafford It was here that he began studying the local flora and soon he became an expert in botany During this time he was compiling notes for his first book, "A Botanical Arrangement of

[1] Roddis and other biographers say that he was a physician with a highly successful practice at Wellington

WILLIAM WITHERING RECEIVING FROM OLD MOTHER HUTTON OF SHROPSHIRE THE RECIPE FOR HER HERB TEA WHICH HAD "RELIEVED AN OXFORD DEAN OF HIS DROPSY"

(Courtesy Parke, Davis and Co)

(It will be noted, on p 238 of this work, that Withering himself does not say that he actually visited or met this old lady, although he does mention Shropshire, and also Dr Cawley, of Oxford, who "had been cured of a Hydrops Pectoris" by foxglove root)

all the Vegetables Naturally Growing in Great Britain'' (London, 1776, v 2) This was a masterful work and went through several editions The author not only gave a description of the plants but also indicated uses to which they might be put and often cited references made to them by the poets

Withering was very popular in Stafford, and it seems that his latent interest in botany had been revived not because of his course in the subject under Hope, a course which had been disagreeable to him, but because of his labors in supplying suitable flowers for a young lady to paint The young lady was his patient, Helena Cook, whom he married in 1772

Although he enjoyed his practice in Stafford and was the only physician at the county infirmary, the practice was a poor one, and counseled by Erasmus Darwin, Withering began the practice of medicine in Birmingham in 1775 At Birmingham, Withering found a congenial circle in the Lunar Society, of which the most eminent members were Joseph Priestley, the great chemist, and James Watt, who perfected the steam engine Withering was very successful in the practice of medicine and soon he was reputed to have the best practice outside of London He aided in the completion of the general hospital at Birmingham and at his own house on stated days he gave the poor free medical advice

Withering's extensive practice caused him to travel day and night While he traveled, he read and wrote On winter nights, to aid him in his studying, he had a light installed in his carriage In this manner he prepared his work, ''An Account of the Scarlet Fever and Sore Throat, or Scarlatina Anginosa'' (London, 1779) Withering also interested himself in chemistry and in mineralogy In 1783 he translated a treatise by Bergman on mineralogy To Withering's discovery of ''Terra Ponderosa'' (the natural barium carbonate), Werner, the German geologist, gave the name ''Witherite'' In 1784 the Royal Society elected Withering a fellow and in 1791 the Linnaean Society similarly honored him (Cushny, p 88)

In 1785, Withering published the little book, ''An Account of the Foxglove,'' which still has the greatest of interest to his profession and which, more than either his botanical or chemical work, entitles him to immortality In this superb monograph on the foxglove (from which we reprint the more important parts) Withering states that his attention was drawn to digitalis in 1775 by the discovery that it was important in the cure of dropsy This remedy was a decoction of herbs which an old woman in his native town, Shropshire, had compounded and used to cure the dropsy in instances in which qualified physicians had failed After carefully analyzing the remedy, Withering found the important ingredient to be foxglove After experimenting with foxglove at great length and satisfying himself as to his results, he prescribed it in his personal dispensary Apparently, his interest was further stimulated by hearing that the drug had been employed successfully in the case of the principal of Brasenose College, Oxford At first he made the leaves of the plant into a decoction, later into an infusion, and sometimes he used the powdered form The use of digitalis quickly spread among Withering's friends in the profession at Birmingham and Edinburgh In 1783 the drug made its appearance in the ''Edinburgh Pharmacopoeia'' Erasmus Darwin, among others, used it (1785) and his son, Charles Darwin, the uncle of the great naturalist, also prescribed it at an early date

On the basis of Withering's description of the patients he treated, it has been assumed that some of his patients suffered from auricular fibrillation But Withering recommended the use of digitalis in dropsy and anasarca only, and was careful to state that it was valueless in the treatment of ovarian cysts and similar conditions His book was a curb on the unqualified uses of digitalis which grew out of the tremendous popularity of the drug Withering did not understand how this drug acted

in dropsy nor did he differentiate its action on cardiac dropsy from its action on other forms of dropsy At the same time he was aware that it exerted some action on the heart and that it retarded the pulse, for he wrote, "That it has a power over the motion of the heart, to a degree yet unobserved in any other medicine, and that this power may be converted to salutary ends"

Besides enjoying a large circle of friends in Birmingham, Withering maintained connections with many of his profession in London His botanical work (ed 2, 1787-1792) served to introduce him to many continental scientists, and the French botanist, L'Heritier de Brutelle, named a genus of plants Witheringia in his honor

In politics Withering was a moderate progressive and held the viewpoint that the constitution under George III required some modifications In spite of this viewpoint and probably because he was sympathetic to the French revolutionists, Withering's house was attacked and he was forced to leave when the home of Priestley was burned in 1791 by a mob He succeeded, however, in carrying off his most precious books and herbariums in wagons camouflaged with straw

Withering suffered from tuberculosis of the lungs which finally undermined his health in 1780 Because of this, he chose to retire from active practice in 1783 From 1790 to 1791 he had repeated attacks of pleurisy and from this time onward his strength declined In 1792 he spent the winter in Lisbon, Portugal, to try the effect of a warmer climate on his health At Lisbon he continued his botanical studies and also made an analysis of the waters of the springs at Caldas da Rainha Withering was not favorably impressed by the climate at Lisbon as a cure for phthisis and felt he had obtained little benefit from his stay He returned there, however, for the winter of 1793

In 1794 his health grew worse, he had inflammatory pulmonary attacks, dyspnea, and repeated hemoptysis His health seemed to improve in 1795, so that he was able to publish in 1796 the third edition of his "Arrangement of British Plants," this time expanded to four volumes From 1797 to 1798 his illness grew worse and he was so dyspneic that even writing was difficult for him He died on October 6, 1799, after twenty-five years of illness He was buried in the old church at Edgbaston In this church is a monument inscribed in his name and encircled with the Witheringia and the purple foxglove of which he wrote

A N

A C C O U N T

O F T H E

F O X G L O V E,

A N D

Some of its Medical Uſes:

W I T H

PRACTICAL REMARKS ON DROPSY, AND OTHER DISEASES

B Y

WILLIAM WITHERING, M D.
Phyſician to the General Hoſpital at Birmingham.

—— *nonumque prematur in annum.*

Horace.

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AN ACCOUNT OF THE FOXGLOVE*

PREFACE

AFTER being frequently urged to write upon this subject, and as often declining to do it, from apprehension of my own inability, I am at length compelled to take up the pen, however unqualified I may still feel myself for the task.

The use of the Foxglove is getting abroad, and it is better the world should derive some instruction, however imperfect, from my experience, than that the lives of men should be hazarded by its unguarded exhibition, or that a medicine of so much efficacy should be condemned and rejected as dangerous and unmanageable.

It is now ten years since I first began to use this medicine. Experience and cautious attention gradually taught me how to use it. For the last two years I have not had occasion to alter the modes of management, but I am still far from thinking them perfect.

It would have been an easy task to have given select cases, whose successful treatment would have spoken strongly in favour of the medicine, and perhaps been flattering to my own reputation. But Truth and Science would condemn the procedure. I have therefore mentioned every case in which I have prescribed the Foxglove, proper or improper, successful or otherwise. Such a conduct will lay me open to the censure of those who are disposed to censure, but it will meet the approbation of others, who are the best qualified to be the judges.

To the Surgeons and Apothecaries, with whom I am connected in practice, both in this town and at a distance, I beg leave to make this public acknowledgement, for the assistance they so readily afforded me, in perfecting some of the cases, and in communicating the events of others.

The ages of the patients are not always exact, nor would the labour of making them so have been repaid by any useful consequences. In a few instances accuracy in that respect was necessary, and there it has been attempted, but in general, an approximation towards the truth, was supposed to be sufficient.

The cases related from my own experience, are generally written in the shortest form I could contrive, in order to save time and labour. Some of them are given more in detail, when particular circumstances made such detail necessary, but the cases communicated by other practitioners, are given in their own words.

*Withering, William. *An Account of the Foxglove*, London, 1785.

I must caution the reader, who is not a practitioner in physic, that no general deductions, decisive upon the failure or success of the medicine, can be drawn from the cases I now present to him These cases must be considered as the most hopeless and deplorable that exist, for physicians are seldom consulted in chronic diseases, till the usual remedies have failed and, indeed, for some years, whilst I was less expert in the management of the Digitalis, I seldom prescribed it, but when the failure of every other method compelled me to do it, so that upon the whole, the instances I am going to adduce, may truly be considered as cases lost to the common run of practice, and only snatched from destruction, by the efficacy of the Digitalis, and this in so remarkable a manner, that, if the properties of that plant had not been discovered, by far the greatest part of these patients must have died

There are men who will hardly admit of anything which an author advances in support of a favourite medicine, and I allow they may have some cause for their hesitation, nor do I expect they will wave their usual modes of judging upon the present occasion I could wish therefore that such readers would pass over what I have said, and attend only to the communications from correspondents, because they cannot be supposed to possess any unjust predilection in favor of the medicine but I cannot advise them to this step, for I am certain they would then close the book, with much higher notions of the efficacy of the plant than they would have learnt from me Not that I want faith in the discernment or in the veracity of my correspondents, for they are men of established reputation, but the cases they have sent me are, with some exceptions, too much selected They are not upon this account less valuable in themselves, but they are not the proper premises from which to draw permanent conclusions

I wish the reader to keep in view, that it is not my intention merely to introduce a new diuretic to his acquaintance, but one which, though not infallible, I believe to be much more certain than any other in present use

After all, in spite of opinion, prejudice, or error, *Time* will fix the real value upon this discovery, and determine whether I have imposed upon myself and others, or contributed to the benefit of science and mankind

Birmingham, 1st July, 1785

INTRODUCTION

The Foxglove is a plant sufficiently common in this island, and as we have but one species, and that so generally known, I should have thought it superfluous either to figure or describe it, had I not more than once seen the leaves of Mullein* gathered for those of the Foxglove On the continent of Europe too, other species are found, and I have been informed

*Verbascum of Linnaeus

that our species is very rare in some parts of Germany, existing only by means of cultivation, in gardens

Our plant is the *Digitalis purpurea*† of Linnaeus It belongs to the 2d order of the 14th class, or the *Didynamia Angiospermia* The *essential characters* of the genus are, *Cup with 5 divisions Blossom bell-shaped, bulging Capsule egg-shaped, 2-celled*—*Linn*

DIGITALIS purpurea Little leaves of the empalement egg-shaped, sharp Blossoms blunt, the upper lip entire *Linn*

I have not observed that any of our cattle eat it The root, the stem, the leaves, and the flowers have a bitter herbaceous taste, but I don't perceive that nauseous bitter which has been attributed to it

This plant ranks amongst the LURIDAE, one of the Linnaean orders in a natural system It has for congenera, *Nicotiana, Atropa, Hyoscyamus, Datura, Solanum,* etc so that from the knowledge we possess of the virtues of those plants, and reasoning from botanical analogy, we might be led to guess at something of its properties

I intended in this place to have traced the history of its effects in diseases from the time of Fuchsius, who first describes it, but I have been anticipated in this intention by my very valuable friend, Dr Stokes of Stourbridge, who has lately sent me the following

HISTORICAL VIEW OF THE PROPERTIES OF DIGITALIS

Fuchsius in his *hist stirp* 1542, is the first author who notices it From him it receives its name of *Digitalis,* in allusion to the German name of *Fingerhut,* which signifies a finger-stall, from the blossoms resembling the finger of a glove

Sensible Qualities Leaves bitterish, very nauseous *Lewis Mat med* I 342

Sensible Effects Some persons, soon after eating of a kind of omolade, into which the leaves of this, with those of several other plants, had entered as an ingredient, found themselves much indisposed, and were presently after attacked with vomitings *Dodonaeus pempt* 170

It is a medicine which is proper only for strong constitutions, as it purges very violently, and excites excessive vomitings *Ray hist* 767

Boerhaave judges it to be of a poisonous nature, *hist plant* but Dr Alston ranks it among those indigenous vegetables, "which, though now disregarded, are medicines of great virtue, and scarcely inferior to any that the Indies afford" *Lewis Mat med* I p 343

Six or seven spoonfuls of the decoction produce nausea and vomiting, and purge, not without some marks of a deleterious quality—*Haller hist* *n* 330 from *Aerial Infl* p 49, 50

†The trivial name *purpurea* is not a very happy one for the blossoms though generally purple, are sometimes of a pure white

The following is an abridged *Account of its Effects upon Turkeys*

M Salerne, a physician at Orleans, having heard that several turkey pouts had been killed by being fed with Foxglove leaves, instead of mullein, he gave some of the same leaves to a large vigorous turkey The bird was so much affected that he could not stand upon his legs, he appeared drunk, and his excrements became reddish Good nourishment restored him to health in eight days

Being then determined to push the experiment further, he chopped some more leaves, mixed them with bran, and gave them to a vigorous turkey cock which weighed seven pounds This bird soon appeared drooping and melancholy, his feathers stared, his neck became pale and retracted The leaves were given him for four days, during which time he took about half a handful These leaves had been gathered about eight days, and the winter was far advanced The excrements, which are naturally green and well formed, became, from the first, liquid and reddish, like those of a dysenteric patient

The animal refusing to eat any more of this mixture which had done him so much mischief, I was obliged to feed him with bran and water only, but notwithstanding this, he continued drooping, and without appetite At times he was seized with convulsions, so strong as to throw him down, in the intervals he walked as if drunk, he did not attempt to perch, he uttered plaintive cries At length he refused all nourishment On the fifth or sixth day the excrements became as white as chalk, afterwards yellow, greenish, and black On the eighteenth day he died, greatly reduced in flesh, for he now weighed only three pounds

On opening him we found the heart, the lungs, the liver, and gall-bladder shrunk and dried up, the stomach was quite empty, but not deprived of its villous coat —*Hist de l'Academ* 1748, p 84

Epilepsy —"It hath beene of later experience found also to be effectual against the falling sicknesse, that divers have been cured thereby, for after the taking of *Decoct manipulor ii c polypod quercin contus ℥iv in cerevisia,* they that have been troubled with it twenty-six years, and have fallen once in a weeke, or two or three times in a moneth, have not fallen once in fourteen or fifteen moneths, that is until the writing hereof "—*Parkinson,* p 654

Scrophula —"The herb bruised, or the juice made up into an ointment, and applied to the place, hath been found by late experience to be availeable for the King's Evill "—*Park* p 654

Several hereditary instances of this disease said to have been cured by it *Aerial Influences,* p 49, 50, quoted by *Haller, hist n* 330

A man with *scrophulous ulcers* in various parts of the body, and which in the right leg were so virulent that its amputation was proposed, cured by *succ express cochl ii bis intra xii dies, in ½ pinta cerevisiae calidae*

The leaves remaining after the pressing out of the juice, were applied every day to the ulcers—*Pract ess* p 40, quoted by *Murray apparat medicam* i p 491

A young woman with a *scrophulous tumour of the eye,* a remarkable *swelling of the upper lip, and painful tumours of the joints of the fingers,* much relieved, but the medicine was left off, on account of its violent effects on the constitution—*Ib* p 42 quoted as above

A man with a *scrophulous tumour of the right elbow,* attended for three years *with excruciating pains,* was nearly cured by four doses of the juice taken once a month—*Ib* p 43 as above

The physicians and surgeons of the Worcester Infirmary have employed it in ointments and poultices with remarkable efficacy—*Ib* p 44 It was recommended to them by Dr Baylies of Evesham, now of Berlin, as a remedy for this disease Dr Wall gave it a tryal, as well externally as internally, but their experiments did not lead them to observe any other properties in it, than those of a highly nauseating medicine and drastic purgative

Wounds In considerable estimation for the healing of all kinds of wounds—*Lobel adv* 245

Principally of use in ulcers, which discharge considerably, being of little advantage in such as are dry—*Hulse,* in R hist 768

Doctor Baylies, physician to his Prussian Majesty, informed me, when at Berlin, that he employed it with great success in caries, and obstinate sore legs

Dyspnoea Pituitosa Sauvages I 657—"Boiled in water, or wine, and drunken doth cut and consume the thicke toughnesse of grosse, and slimie flegme, and naughtie humours The same, or boiled with honied water or sugar, doth scoure and clense the brest, ripeneth and bring foorth tough and clammie flegme It openeth also the stoppage of the liver spleene and milt, and of the inwarde parts"—*Gerarde hist* ed 1, p 647

"Whensoever there is need of a rarefying or extenuating of tough flegme or viscous humours troubling the chest,—the decoction or juice hereof made up with sugar or honey is availeable, as also to clense and purge the body both upwards and downwards sometimes, of tough flegme, and clammy humours, notwithstanding that these qualities are found to bee in it, there are but few physitions in our times that put it to these uses, but it is in a manner wholly neglected"—*Parkinson,* p 654

Previous to the year 1777, you informed me of the great success you had met with in curing dropsies by means of the fol Digitalis, which you then considered as a more certain diuretic than any you had ever tried Sometime afterwards, Mr Russel, surgeon, of Worcester, having heard of the success which had attended some cases in which you had given it, requested

me to obtain for him any information you might be inclined to communicate respecting its use In consequence of this application, you wrote to me in the following terms

In a letter which I received from you in London, dated September 29, 1778, you write as follows —"I wish it was as easy to write upon the Digitalis—I despair of pleasing myself or instructing others, in a subject so difficult It is much easier to write upon a disease than upon a remedy The former is in the hands of nature, and a faithful observer, with an eye of tolerable judgement, cannot fail to delineate a likeness The latter will ever be subject to the whims, the inaccuracies, and the blunders of mankind "

In my notes I find the following memorandum—"February 20th, 1779, gave an account of Doctor Withering's practice, with the precautions necessary to its success, to the Medical Society at Edinburgh "—In the course of that year, the Digitalis was prescribed in the Edinburgh Infirmary, by Dr Hope, and in the following year, whilst I was Clerk to Dr Home, as Clinical Professor, I had a favourable opportunity of observing its sensible effects

In one case in which it was given properly at first, the urine began to flow freely on the second day On the third, the swellings began to subside The dose was then increased more than *quadruple* in the twenty-four hours On the fifth day sickness came on, and much purging, but the urine still increased though the pulse sunk to 50 On the 7th day, a *quadruple* dose of the infusion was ordered to be taken every third hour, so as to bring on nausea again The pulse fell to forty-four, and at length to thirty-five in a minute The patient gradually sunk and died on the sixteenth day, but previous to her death, for two or three days, her pulse rose to near one hundred —It is needless to observe to you, how widely the treatment of this case differed from the method which you have found so successful

AN ACCOUNT OF THE INTRODUCTION OF FOXGLOVE INTO MODERN PRACTICE

As the more obvious and sensible properties of plants, such as colour, taste, and smell, have but little connexion with the diseases they are adapted to cure, so their peculiar qualities have no certain dependence upon their external configuration Their chemical examination by fire, after an immense waste of time and labour, having been found useless, is now abandoned by general consent Possibly other modes of analysis will be found out, which may turn to better account but we have hitherto made only a very small progress in the chemistry of animal and vegetable substances Their virtues must therefore be learnt either from

observing their effects upon insects and quadrupeds, from analogy, deduced from the already known powers of some of their congenera, or from the empirical usages and experience of the populace

The first method has not yet been much attended to, and the second can only be perfected in proportion as we approach towards the discovery of a truly natural system, but the last, as far as it extends, lies within the reach of every one who is open to information, regardless of the source from whence it springs

It was a circumstance of this kind which first fixed my attention on the Foxglove

In the year 1775, my opinion was asked concerning a family receipt for the cure of the dropsy I was told that it had long been kept a secret by an old woman in Shropshire, who had sometimes made cures after the more regular practitioners had failed I was informed also, that the effects produced were violent vomiting and purging, for the diuretic effects seemed to have been overlooked This medicine was composed of twenty or more different herbs, but it was not very difficult for one conversant in these subjects, to perceive, that the active herb could be no other than Foxglove

My worthy predecessor in this place, the very humane and ingenious Dr Small, had made it a practice to give his advice to the poor during one hour in a day This practice, which I continued until we had an Hospital opened for the reception of the sick poor, gave me an opportunity of putting my ideas into execution in a variety of cases, for the number of poor who thus applied for advice, amounted to between two and three thousand annually I soon found the Foxglove to be a very powerful diuretic, but then, and for a considerable time afterwards, I gave it in doses very much too large, and urged its continuance too long, for misled by reasoning from the effects of the squill, which generally acts best upon the kidneys when it excites nausea, I wished to produce the same effect by the Foxglove In this mode of prescribing, when I had so many patients to attend to in the space of one or at most of two hours, it will not be expected that I could be very particular, much less could I take notes of all the cases which occurred Two or three of them only, in which the medicine succeeded, I find mentioned amongst my papers It was from this kind of experience that I ventured to assert, in the Botanical Arrangement published in the course of the following spring, that the Digitalis purpurea "merited more attention than modern practice bestowed upon it"

I had not, however, yet introduced it into the more regular mode of prescription, but a circumstance happened which accelerated that event My truly valuable and respectable friend, Dr Ash, informed me that Dr Cawley, then principal of Brazen Nose College, Oxford, had been cured of a Hydrops Pectoris, by an empirical exhibition of the root of the Fox-

glove, after some of the first physicians of the age had declared they could do no more for him. I was now determined to pursue my former ideas more vigorously than before, but was too well aware of the uncertainty which must attend on the exhibition of the *root* of a *biennial* plant, and therefore continued to use the *leaves*. These I had found to vary much as to dose, at different seasons of the year; but I expected, if gathered always in one condition of the plant, viz. when it was in its flowering state, and carefully dried, that the dose might be ascertained as exactly as that of any other medicine; nor have I been disappointed in this expectation. The more I saw of the great powers of this plant, the more it seemed necessary to bring the doses of it to the greatest possible accuracy. I suspected that this degree of accuracy was not reconcileable with the use of a *decoction*, as it depended not only upon the care of those who had the preparation of it, but it was easy to conceive from the analogy of another plant of the same natural order, the tobacco, that its active properties might be impaired by long boiling. The decoction was therefore discarded, and the *infusion* substituted in its place. After this I began to use the leaves in *powder*, but I still very often prescribe the infusion.

Further experience convinced me, that the *diuretic* effects of this medicine do not at all depend upon its exciting a nausea or vomiting; but, on the contrary, that though the increased secretion of urine will frequently succeed to, or exist along with these circumstances, yet they are so far from being friendly or necessary, that I have often known the discharge of urine checked, when the doses have been imprudently urged so as to occasion sickness.

If the medicine purges, it is almost certain to fail in its desired effect; but this having been the case, I have seen it afterwards succeed when joined with small doses of opium, so as to restrain its action on the bowels.

In the summer of the year 1776, I ordered a quantity of the leaves to be dried, and as it then became possible to ascertain its doses, it was gradually adopted by the medical practitioners in the circle of my acquaintance.

In the month of November 1777, in consequence of an application from that very celebrated surgeon, Mr. Russel, of Worcester, I sent him the following account, which I choose to introduce here, as showing the ideas I then entertained of the medicine, and how much I was mistaken as to its real dose.—"I generally order it in decoction. Three drams of the dried leaves, collected at the time of the blossoms expanding, boiled in twelve to eight ounces of water. Two spoonfuls of this medicine, given every two hours, will sooner or later excite a nausea. I have sometimes used the green leaves gathered in winter, but then I order three times the weight, and in one instance I used three ounces to a pint decoction, before the desired effect took place. I considered the Foxglove thus given, as the most certain diuretic I know, nor do its diuretic effects depend merely upon the nausea it produces, for in cases where squill and ipecac have

been so given as to keep up a nausea several days together, and the flow of urine not taken place, I have found the Foxglove to succeed, and I have, in more than one instance, given the Foxglove in smaller and more distant doses, so that the flow of urine has taken place without any sensible affection of the stomach, but in general I give it in the manner first mentioned, and order one dose to be taken after the sickness commences. I then omit all medicines, except those of the cordial kind are wanted, during the space of three, four, or five days. By this time the nausea abates, and the appetite becomes better than it was before. Sometimes the brain is considerably affected by the medicine, and indistinct vision ensues, but I have never yet found any permanent bad effects from it."

"I use it in the Ascites, Anasarca, and Hydrops Pectoris, and so far as the removal of the water will contribute to cure the patient, so far may be expected from this medicine: but I wish it not to be tried in ascites of female patients, believing that many of these cases are dropsies of the ovaria, and no sensible man will ever expect to see these encysted fluids removed by any medicine."

"I have often been obliged to evacuate the water repeatedly in the same patient, by repeating the decoction, but then this has been at such distance of time as to allow of the interference of other medicines and a proper regimen, so that the patient obtains in the end a perfect cure. In these cases the decoction becomes at length so very disagreeable, that a much smaller quantity will produce the effect, and I often find it necessary to alter its taste by the addition of Aq. Cinnam. sp. or Aq. Juniper. composita."

"I allow, and indeed enjoin my patients to drink very plentifully of small liquors through the whole course of the cure, and sometimes, where the evacuations have been very sudden, I have found a bandage as necessary as in the use of the trochar."

Early in the year 1779, a number of dropsical cases offered themselves to my attention, the consequences of the scarlet fever and sore throat which had raged so very generally amongst us in the preceding year. Some of these had been cured by squills or other diuretics, and relapsed, in others, the dropsy did not appear for several weeks after the original disease had ceased: but I am not able to mention many particulars, having omitted to make notes. This, however, is the less to be regretted, as the symptoms in all were very much alike, and they were all without an exception cured by the Foxglove.

This last circumstance encouraged me to use the medicine more frequently than I had done heretofore, and the increase of practice had taught me to improve the management of it.

In February 1779, my friend, Dr. Stokes, communicated to the Medical Society at Edinburgh the result of my experience of the Foxglove, and, in a letter addressed to me in November following, he says, "Dr. Hope,

in consequence of my mentioning its use to my friend, Dr Broughton, has tried the Foxglove in the Infirmary with success " Dr Stokes also tells me that Dr Hamilton cured dropsies with it in the year 1781

I am informed by my very worthy friend Dr Duncan, that Dr Hamilton, who learnt its use from Dr Hope, has employed it very frequently in the Hospital at Edinburgh Dr Duncan also tells me, that the late very ingenious and accomplished Mr Charles Darwin, informed him of its being used by his father and myself, in cases of Hydrothorax, and that he has ever since mentioned it in his lectures, and sometimes employed it in his practice

At length, in the year 1783, it appeared in the new edition of the Edinburgh Pharmacopoeia, into which, I am told, it was received in consequence of the recommendation of Dr Hope But from which, I am satisfied, it will be again very soon rejected, if it should continue to be exhibited in the unrestrained manner in which it has heretofore been used at Edinburgh, and in the enormous doses in which it is now directed in London

In the following cases the reader will find other diseases besides dropsies, particularly several cases of consumption I was induced to try it in these, from being told, that it was much used in the West of England, in the Phthisis Pulmonalis, by the common people In this disease, however, in my hands, it has done but little service, and yet I am disposed to wish it a further trial, for in a copy of Parkinson's Herbal, which I saw about two years ago, I found the following manuscript note at the article Digitalis, written I believe, by a Mr Saunders, who practiced for many years with great reputation as a surgeon and apothecary at Stourbridge, in Worcestershire

"Consumptions are cured infallibly by weak decoction of Foxglove leaves in water, or wine and water, and drank for constant drink Or take of the juice of the herb and flowers, clarify it, and make a fine syrup with honey, of which take three spoonfuls thrice in a day, at physical hours The use of these two things of late has done, in consumptive cases, great wonders But be cautious of its use, for it is of a vomiting nature In these things begin sparingly, and increase the dose as the patient's strength will bear, least, instead of a sovereign medicine, you do real damage by this infusion or syrup "

The precautions annexed to his encomiums of this medicine, lead one to think that he has spoken from his own proper experience

I have lately been told, that a person in the neighborhood of Warwick, possesses a famous family receipt for the dropsy, in which the Foxglove is the active medicine, and a lady from the western part of Yorkshire assures me, that the people in her country often cure themselves of dropsical complaints by drinking Foxglove tea In confirmation of this, I recollect about two years ago being desired to visit a travelling York-

shire tradesman I found him incessantly vomiting, his vision indistinct, his pulse forty in a minute Upon enquiry it came out, that his wife had stewed a large handful of green Foxglove leaves in a half a pint of water, and given him the liquor, which he drank at one draught, in order to cure him of an asthmatic affection This good woman knew the medicine of her country, but not the dose of it, for her husband narrowly escaped with his life

It is probable that this rude mode of exhibiting the Foxglove has been more general than I am at present aware of, but it is wonderful that no author seems to have been acquainted with its effects as a diuretic

OF THE PREPARATIONS AND DOSES OF THE FOXGLOVE

Every part of the plant has more or less the same bitter taste, varying, however, as to strength, and changing with the age of the plant and the season of the year

ROOT —This varies greatly with the age of the plant When the stem has shot up for flowering, which it does the second year of its growth, the root becomes dry, nearly tasteless, and inert

Some practitioners, who have used the root, and been so happy as to cure their patients without exciting sickness, have been pleased to communicate the circumstance to me as an improvement in the use of the plant I have no doubt of the truth of their remarks, and I thank them But the case of Dr Cawley puts this matter beyond dispute The fact is, they have fortunately happened to use the root in its approach to its inert state, and consequently have not over dosed their patients I could, if necessary, bring other proof to shew that the root is just as capable as the leaves, of exciting nausea

STEM —The stem has more taste than the root has, in the season the stem shoots out, and less taste than the leaves I do not know that it has been particularly selected for use

LEAVES —These vary greatly in their efficacy at different seasons of the year, and, perhaps, at different stages of their growth, but I am not certain that this variation keeps pace with the greater or lesser intensity of their bitter taste

Some who have been habituated to the use of the recent leaves, tell me, that they answer their purpose at every season of the year, and I believe them, notwithstanding I myself have found very great variations in this respect The solution of this difficulty is obvious They have used the leaves in such large proportion, that the doses have been sufficient, or more than sufficient, even in their most inefficacious state *The leaf-stalks* seem, in their sensible properties, to partake of an intermediate state between the leaves and the stem

FLOWERS —The petals, the chives, and the pointal have nearly the taste of the leaves, and it has been suggested to me, by a very sensible and judicious friend, that it might be well to fix on the flower for internal use I see no objection to the proposition, but I have not tried it

SEEDS —These I believe are equally untried

From this view of the different parts of the plant, it is sufficiently obvious why I still continue to prefer the leaves

These should be gathered after the flowering stem has shot up, and about the time that the blossoms are coming forth

The leaf-stalk and mid-rib of the leaves should be rejected, and the remaining part should be dried, either in the sun-shine, or on a tin pan or pewter dish before a fire

If well dried, they readily rub down to a beautiful green powder, which weighs something less than one-fifth of the original weight of the leaves Care must be taken that the leaves be not scorched in drying, and they should not be dried more than what is requisite to allow of their being readily reduced to powder

I give to adults, from one to three grains of this powder twice a day In the reduced state in which physicians generally find dropsical patients, four grains a day are sufficient I sometimes give the powder alone, sometimes unite it with aromatics, and sometimes form it into pills with a sufficient quantity of soap or gum ammoniac

If a liquid medicine be preferred, I order a dram of these dried leaves to be infused for four hours in half a pint of boiling water, adding to the strained liquor an ounce of any spiritous water One ounce of this infusion given twice a day, is a medium dose for an adult patient If the patient be stronger than usual, or the symptoms very urgent, this dose may be given once in eight hours, and on the contrary in many instances half an ounce at a time will be quite sufficient About thirty grains of the powder or eight ounces of the infusion, may generally be taken before the nausea commences

The ingenuity of man has ever been fond of exerting itself to vary the forms and combinations of medicines Hence we have spirituous, vinous, and acetous tinctures, extracts hard and soft, syrups with sugar or honey, etc but the more we multiply the forms of any medicine, the longer we shall be in ascertaining its real dose I have no lasting objection however to any of these formulae except the extract, which, from the nature of its preparation must ever be uncertain in its effects, and a medicine whose fullest dose in substance does not exceed three grains, cannot be supposed to stand in need of condensation

It appears from several of the cases, that when the Digitalis is disposed to purge, opium may be joined with it advantageously, and when the bowels are too tardy, jalap may be given at the same time without interfering with its diuretic effects, but I have not found benefit from any other adjunct

From this view of the doses in which the Digitalis really ought to be exhibited, and from the evidence of many of the cases, in which it appears to have been given in quantities six, eight, ten or even twelve times more than necessary, we must admit as an inference either that this medicine is perfectly safe when given as I advise, or that the medicines in daily use are highly dangerous

EFFECTS, RULES AND CAUTIONS

The Foxglove when given in very large and quickly repeated doses, occasions sickness, vomiting, purging, giddiness, confused vision, objects appearing green or yellow, increased secretion of urine, with frequent motions to part with it, and sometimes inability to retain it, slow pulse, even as slow as 35 in a minute, cold sweats, convulsions, syncope, death *

When given in a less violent manner, it produces most of these effects in a lower degree, and it is curious to observe, that the sickness, with a certain dose of the medicine, does not take place for many hours after its exhibition has been discontinued, that the flow of urine will often precede, sometimes accompany, frequently follow the sickness at the distance of some days, and not infrequently be checked by it The sickness thus excited, is extremely different from that occasioned by any other medicine, it is peculiarly distressing to the patient, it ceases, it recurs again as violent as before, and thus it will continue to recur for three or four days, at distant and more distant intervals

These sufferings of the patient are generally rewarded by a return of appetite, much greater than what existed before the taking of the medicine

But these sufferings are not at all necessary, they are the effects of our inexperience, and would in similar circumstances, more or less attend the exhibition of almost every active and powerful medicine we use

Perhaps the reader will better understand how it ought to be given, from the following detail of my own improvement, than from precepts peremptorily delivered, and their source veiled in obscurity

At first I thought it necessary *to bring on and continue the sickness in order to ensure the diuretic effects*

I soon learnt that the nausea being once excited, it was unnecessary to repeat the medicine, as it was certain to recur frequently, at intervals more or less distant

Therefore my patients were ordered *to persist until the nausea came on, and then to stop* But it soon appeared that the diuretic effects would often take place first, and sometimes be checked when the sickness or a purging supervened

The direction was therefore enlarged thus—*Continue the medicine until the urine flows, or sickness or purging takes place*

*I am doubtful whether it does not sometimes excite a copious flow of saliva

I found myself safe under this regulation for two or three years, but at length cases occurred in which the pulse would be retarded to an alarming degree, without any other preceding effect

The directions therefore required an additional attention to the state of the pulse, and it was moreover of consequence not to repeat the doses too quickly, but to allow sufficient time for the effects of each to take place, as it was found very possible to pour in an injurious quantity of the medicine, before any of the signals for forbearance appeared

Let the medicine therefore be given in the doses, and at the intervals mentioned above —let it be continued until it either acts on the kidneys, the stomach, the pulse, or the bowels, let it be stopped upon the first appearance of any one of these effects, and I will maintain that the patient will not suffer from its exhibition, nor the practitioner be disappointed in any reasonable expectation

If it purges, it seldom succeeds well

The patient should be enjoined to drink very freely during its operation I mean, they should drink whatever they prefer, and in as great quantity as their appetite for drink demands This direction is the more necessary, as they are very generally prepossessed with an idea of drying up a dropsy, by abstinence from liquids, and fear to add to the disease, by indulging their inclination to drink

In cases of ascites and anasarca, when the patients are weak, and the evacuation of the water rapid, the use of proper bandage is indispensably necessary to their safety

If the water should not be wholly evacuated, it is best to allow an interval of several days before the medicine be repeated, that food and tonics may be administered, but truth compels me to say, that the usual tonic medicines have in these cases very often deceived my expectations

From some cases which have occurred in the course of the present year, I am disposed to believe that the Digitalis may be given in small doses, viz two or three grains a day, so as gradually to remove a dropsy, without any other than mild diuretic effects, and without any interruption to its use until the cure be completed

If inadvertently the doses of the Foxglove should be prescribed too largely, exhibited too rapidly, or urged to too great a length, the knowledge of a remedy to counteract its effects would be a desirable thing Such a remedy may perhaps in time be discovered The usual cordials and volatiles are generally rejected from the stomach, aromatics and strong bitters are longer retained, brandy will sometimes remove the sickness when only slight, I have sometimes thought small doses of opium useful, but I am more confident of the advantage from blisters Mr Jones in one case, found mint tea to be retained longer than other things

CONSTITUTION OF PATIENTS

Independent of the degree of disease, or of the strength or age of the patient, I have had occasion to remark, that there are certain constitutions favourable, and others unfavourable to the success of the Digitalis

From large experience, and attentive observation, I am pretty well enabled to decide *a priori* upon this matter, and I wish to enable others to do the same but I feel myself hardly equal to the undertaking The following hints, however, aiding a degree of experience in others, may lead them to accomplish what I yet can describe but imperfectly

It seldom succeeds in men of great natural strength, of tense fibre, of warm skin, of florid complexion, or in those with a tight and cordy pulse

If the belly in ascites be tense, hard, and circumscribed, or the limbs in anasarca solid and resisting, we have but little to hope

On the contrary, if the pulse be feeble or intermitting, the countenance pale, the lips livid, the skin cold, the swollen belly soft and fluctuating, or the anasarcous limbs readily pitting under the pressure of the finger, we may expect the diuretic effects to follow in a kindly manner

In cases which foil every attempt at relief, I have been aiming, for some time past, to make such a change in the constitution of the patient, as might give a chance of success to the Digitalis

By blood-letting, by neutral salts, by crystals of tartar, squills and occasional purging, I have succeeded, though imperfectly Next to the use of the lancet, I think nothing lowers the tone of the system more effectually than the squill, and consequently it will always be proper, in such cases, to use the squill, for if that fail in its desired effect, it is one of the best preparatives to the adoption of the Digitalis

A tendency to paralytic affections, or a stroke of the palsy having actually taken place, is no objection to the use of the Digitalis, neither does a stone existing in the bladder forbid its use Theoretical ideas of sedative effects in the former, and apprehensions of its excitement of the urinary organs in the latter case, might operate so as to make us withhold relief from the patient, but experience tells me, that such apprehensions are groundless

INFERENCES

To prevent any improper influence, which the above recitals of the efficacy of the medicine, aided by the novelty of the subject, may have upon the minds of the younger part of my readers, in raising their expectations to too high a pitch, I beg leave to deduce a few inferences, which I apprehend the facts will fairly support

I That the Digitalis will not universally act as a diuretic

II That it does do so more generally than any other medicine

III That it will often produce this effect after every other probable method has been fruitlessly tried

IV That if this fails, there is but little chance of any other medicine succeeding

V That in proper doses, and under the management now pointed out, it is mild in its operation, and gives less disturbance to the system than squill, or almost any other active medicine

VI That when dropsy is attended by palsy, unsound viscera, great debility, or other complication or disease, neither the Digitalis, nor any other diuretic can do more than obtain a truce to the urgency of the symptoms, unless by gaining time, it may afford opportunity for other medicines to combat and subdue the original disease

VII That the Digitalis may be used with advantage in every species of dropsy, except the encysted

VIII That it may be made subservient to the cure of diseases, unconnected with dropsy

IX That it has a power over the motion of the heart, to a degree yet unobserved in any other medicine, and that this power may be converted to salutary ends

PRACTICAL REMARKS ON DROPSY, AND SOME OTHER DISEASES

The following remarks consist partly of matter of fact, and partly of opinion The former will be permanent, the latter must vary with the detection of error, or the improvement of knowledge I hazard them with diffidence, and hope they will be examined with candour, not by a contrast with other opinions, but by an attentive comparison with the phenomena of disease

Anasarca

1 The anasarca is generally curable when seated in the sub-cutaneous cellular membrane, or in the substance of the lungs

2 When the abdominal viscera in general are greatly enlarged, which they sometimes are, without effused fluid in the cavity of the abdomen, the disease is incurable After death, the more solid viscera are found very large and pale If the cavity contains water, that water may be removed by diuretics

3 In swollen legs and thighs, where the resistance to pressure is considerable, the tendency to transparency in the skin not obvious, and where the alteration of posture occasions but little alteration in the state of distention, the cure cannot be effected by diuretics

Is this difficulty of cure occasioned by spissitude in the effused fluids, by want of proper communication from cell to cell, or is the disease rather caused by a morbid growth of the solids, than by an accumulation of fluid?

Is not this disease in the limbs similar to that of the viscera (2)?

4 Anasarcous swellings often take place in palsied limbs, in arms as well as legs, so that the swelling does not depend merely upon position

5 Is there not cause to suspect that many dropsies originate from *paralytic affections of the lymphatic absorbents*? And if so, is it not probable that the Digitalis, which is so effectual in removing dropsy, may also be used advantageously in some kinds of palsy?

Ascites

6 If existing alone (*i e*) without accompanying anasarca, is in children curable, in adults generally incurable by medicines Tapping may be used here with better chance for success than in more complicated dropsies Sometimes cured by vomiting

Ascites and Anasarca

7 Incurable if dependent *upon irremediably diseased viscera, or on a* gouty constitution, so debilitated, that the gouty paroxysms no longer continue to be formed

In every other situation the disease yields to diuretics and tonics

Ascites, Anasarca, and Hydrothorax

8 Under this complication, though the symptoms admit of relief, the restoration of the constitution can hardly be hoped for

Asthma

9 The true spasmodic asthma, a rare disease—is not relieved by Digitalis

10 In the greater part of what are called asthmatical cases, the real disease is *anasarca of the lungs,* and is *generally to be cured by diuretics* (See 1) This is almost always combined with some swelling of the legs

11 There is another kind of asthma, in which change of posture does not much affect the patient I believe it to be caused by an infarction of the lungs It is incurable by diuretics, but it is often accompanied with a degree of anasarca, and so far it admits of relief

Is not this disease similar to that in the limbs at (3) and also to that of the abdominal viscera at (2)?

Asthma and Anasarca

12 If the asthma be of the kind mentioned at (9 and 11) diuretics can only remove the accompanying anasarca But if the affection of the breath depends also upon cellular effusion, as it mostly does, the patient may be taught to expect a recovery

Asthma and Ascites

13 A rare combination, but not incurable if the abdominal viscera are sound The asthma is here most probably of the anasarcous kind (10),

and this being seldom confined to the lungs only, the disease generally appears in the following form

Asthma, Ascites, and Anasarca

14 The curability of this combination will depend upon the circumstances mentioned in the preceding section, taking also into the account the strength or weakness of the patient

Epilepsy

15 In epilepsy dependent upon effusion, the Digitalis will effect a cure, and in the cases alluded to, the dropsical symptoms were unequivocal It has not had a sufficient trial in my hands, to determine what it can do in other kinds of epilepsy

Hydatid Dropsy

16 This may be distinguished from common ascites, by the want of evident fluctuation It is common to both sexes It does not admit of a cure either by tapping or by medicine

Hydrocephalus

17 This disease, which has of late so much attracted the attention of the medical world, I believe, originates in inflammation, and that the water found in the ventricles of the brain after death, is the consequence, and not the cause of the illness

It has seldom happened to me to be called upon in the earlier stages of this complaint, and the symptoms are at first so similar to those usually attendant upon dentition and worms, that it is very difficult to pronounce decidedly upon the real nature of the disease, and it is rather from the failure of the usual modes of relief, than from any other more decided observation, that we at length dare to give it a name

Hydrothorax

18 Under this name I also include the dropsy of the pericardium The intermitting pulse, and pain in the arms, sufficiently distinguish this disease from asthma, and from anasarcous lungs

It is very universally cured by the Digitalis

19 I lately met with two cases which had been considered and treated as angina pectoris They both appeared to me to be cases of hydrothorax One subject was a clergyman, whose strength had been so compleatly exhausted by the continuance of the disease, and the attempts to relieve it, that he did not survive many days The other was a lady, whose time of life made me suspect effusion I directed her to take small doses of the pulv Digitalis, which in eight days removed all her complaints This happened six months ago, and she remains perfectly well

Hydrothorax and Anasarca

20 This combination is very frequent, and, I believe, may always be cured by the Digitalis

21 Dropsies in the chest either with or without anasarcous limbs, are much more curable than those of the belly Probably because the abdominal viscera are more frequently diseased in the latter than in the former cases

Insanity

22 I apprehend this disease to be more frequently connected with serous effusion than has been commonly imagined

Nephritis Calculosa

24 We have had sufficient evidence of the efficacy of the Foxglove in removing the Dysuria and other symptoms of this disease, but probably it is not in these cases preferable to the tobacco *

Ovarium Dropsy

25 This species of encysted dropsy is not without difficulty distinguishable from an ascites, and yet it is necessary to distinguish them, because the two diseases require different treatment and because the probability of a cure is much greater in one than in the other

26 The ovarium dropsy is generally slow in its progress, for a considerable time the patient though somewhat emaciated, does not lose the appearance of health, and the urine flows in the usual quantity It is seldom that the practitioner is called in early enough to distinguish by the feel on which side the cyst originated, and the patients do not attend to that circumstance themselves They generally menstruate regularly in the incipient state of the disease, and it is not until the pressure from the sac becomes very great, that the urinary secretion diminishes In this species of dropsy, the patients, upon being questioned, acknowledge even from a pretty early date, pains in the upper and inner parts of the thighs, similar to those which women experience in a state of pregnancy These pains are for a length of time greater in one thigh than in the other, and I believe it will be found that the disease originated on that side

27 The ovarium dropsy defies the power of medicine It admits of relief, and sometimes of a cure, by tapping I submit to the consideration of practitioners, how far we may hope to cure this disease by a seton or a caustic

28 When tapping becomes necessary, I always advise the adoption of the waist coat bandage or belt, invented by the late very justly celebrated

*See an original and valuable treatise by Dr Fowler, entitled, *Medical Reports of the Effects of Tobacco*

Dr Monro, and described in the first volume of the Medical Essays I also enjoin my patients to wear this bandage afterwards, from a persuasion that it retards the return of the disease The proper use of bandage, when the disorder first discovers itself, certainly contributes much to prevent its increase

Ovarium Dropsy With Anasarca

29 The anasarca does not appear until the encysted dropsy is very far advanced It is then probably caused by weakness and pressure The Digitalis removes it for a time

Phthisis Pulmonalis

30 This is a very increasing malady in the present day It is no longer limited to the middle part of life children at five years of age die of it, and old people at sixty or seventy It is not confined to the flat-chested, the fair-skinned, the blue-eyed, the light-haired, or the scrophulous it often attacks people with full chests, brown skins, dark hair and eyes, and those in whose family no scrophulous taint can be traced It is certainly infectious The very strict laws still existing in Italy to prevent the infection from consumptive patients, were probably not enacted originally without a sufficient cause We seem to be approaching to that state which first made such restrictions necessary, and in the further course of time, the disease will probably fall off again, both in virulency and frequency

31 The younger part of the female sex are liable to a disease very much resembling a true consumption, and from which it is difficult to distinguish it, but this disease is curable by steel and bitters A criterion of true phthisis has been sought for in the state of the teeth, but the exceptions to that rule are numerous An unusual dilatation of the pupil of the eye, is the most certain characteristic

32 Sydenham asserts, that the bark did not more certainly cure an intermittent, than riding did a consumption We must not deny the truth of an assertion, from such authority, but we must conclude that the disease was more easily curable a century ago than it is at present

33 If the Digitalis is no longer useful in consumptive cases, it must be that I know not how to manage it, or that the disease is more fatal than formerly, for it would be hard to deny the testimony cited at [page 241] I wish others would undertake the enquiry

34 When phthisis is accompanied with anasarca, or when there is reason to suspect hydrothorax, the Digitalis will often relieve the sufferings, and prolong the life of the patient

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*Many years ago I communicated to my friend, Dr Percival, an account of some trials of breathing fixed air in consumptive cases The results were published by him in the second Vol of his very useful *Essays Medical and Experimental*, and have since been copied into other publications I take this opportunity of acknowledging that I suspect myself to have been mistaken in the nature of the disease there mentioned to have been cured I believe it was a case of *Vomica*, and not a true *Phthisis* that was cured The Vomica is almost always curable The fixed air corrects the smell of the matter and very shortly removes the hectic fever My patients not only inspire it but I keep large jars of the effervescing mixture constantly at work in their chambers

35 Many years ago, during an attendance upon Mr B----, of a consumptive family, and himself in the last stage of a phthisis, after he was so ill as to be confined to his chamber, his breathing became so extremely difficult and distressing, that he wished rather to die than to live, and urged me warmly to devise some mode to relieve him Suspecting serous effusion to be the cause of his symptoms, and he being a man of sense and resolution, I fully explained my ideas to him, and told him what kind of operation might afford him a chance of relief, for I was then but little acquainted with the Digitalis He was earnest for the operation to be tried, and with the assistance of Mr Parrott, a very respectable surgeon of this place, I got an opening made between the ribs upon the lower and hinder part of the thorax About a pint of fluid was immediately discharged, and his breath became easy This fluid coagulated by heat

After some days a copious purulent discharge issued from the opening, his cough became less troublesome, his expectoration less copious, his appetite and strength returned, he got abroad, and the wound, which became very troublesome, was allowed to heal

He then undertook a journey to London, whilst there he became worse returned home, and died consumptive some weeks afterwards

Puerperal Anasarca

36 This disease admits of an easy and certain cure by the Digitalis

37 This species of dropsy may originate from other causes than child birth In the beginning of last March, a gentleman at Wolverhampton desired my advice for very large and painful swelled legs and thighs He was a temperate man, not of a dropsical habit, had great pain in his groins, and attributed his complaints to a fall from his horse He had taken diuretics, and the strongest drastic purgatives with very little benefit Considering the anasarca as caused by the diseased inguinal glands, I ordered a common poultice and mercurial ointment to the groins, three grains of pulv fol Digitalis night and morning, and a cooling diuretic decoction in the day-time He soon lost his pain, and the swellings gradually subsided

THE END

1788

MATTHEW BAILLIE

DESCRIPTION OF A CASE OF CONGENITAL DEXTROCARDIA WITH COMPLETE SITUS-TRANSVERSUS

MATTHEW BAILLIE

From the portrait collection of Northwestern University Medical School

(Courtesy Petrolagar Laboratories)

MATTHEW BAILLIE

(1761-1823)

IT IS of interest to note that Baillie wrote of the pertinent events concerning his own life, and the manuscript, in his own handwriting, is now in the Library of the Royal College of Surgeons of England In 1896 it was printed in the "Practitioner" This illuminating account forms the basis for our brief discussion

Matthew Baillie was born in the Manse of Shots and County of Lanark, Scotland, on October 27, 1761 His father, the Reverend Dr James Baillie was, according to his son, "a man of the most respectable character" He ultimately became professor of divinity in the University of Glasgow

Dorothea Baillie, the mother of Matthew, was a sister of William and John Hunter, the celebrated surgeons and anatomists, thus, early in life, Baillie received much genuine encouragement for his career in medicine

Baillie's early education was obtained at Hamilton There he studied first at the English School and later at the Latin School At the age of thirteen, he became a student at the University of Glasgow There he continued his study of Greek and Latin for a period of five years, receiving a thorough training in the classics and in philosophy

In 1779, Baillie, acting on the advice of his uncle, Dr William Hunter, decided to enter the profession of medicine However, he had been appointed to an exhibition in Balliol College, Oxford, and for the next eighteen months he remained there, improving himself in the classics

In 1780 he went to London to live with his uncle, Dr William Hunter, and to attend his lectures in anatomy and dissections at the Great Windmill Street School During the succeeding years he also studied chemistry, materia medica, and medicine under Dr George Fordyce (1736-1802), who was, for several years, the most popular lecturer in London on these subjects Baillie also attended the lectures on surgery given by his other uncle, John Hunter, and the lectures on midwifery which were given jointly by Dr Thomas Denman (1733-1815) and Dr William Osborne (1736-1808)

Dr William Hunter died in March, 1783 He left Baillie a legacy of 5,000 pounds and a small estate in Scotland Baillie, believing that his uncle, John Hunter, had a better claim to the estate, ceded it to him Dr Hunter had arranged through his will that Baillie should succeed him as lecturer in anatomy, in association with William Cumberland Cruikshank (1745-1800) At the Great Windmill Street School Baillie continued as lecturer in anatomy for fifteen years, in spite of the irascibility of his associate Meanwhile, his private practice had increased and in 1799 he relinquished his anatomic lectures

Shortly after his uncle's death, Baillie received the degree of Bachelor of Arts from Oxford University He also received the degree of Bachelor of Medicine in 1786 and that of Doctor of Medicine in 1789, both from Oxford University

In 1787 he was appointed physician at St George's Hospital and in 1790 he received his fellowship in the Royal College of Physicians, London

Baillie, in 1791, was married to Sophia, the youngest of twin daughters of Dr. Thomas Denman, under whom he had studied obstetrics. This marriage resulted in the birth of three children: James, Elizabeth Margaret, and William Hunter Baillie.

One of Baillie's patients was the celebrated Dr. David Pitcairn (1749-1809). When Pitcairn retired from the practice of medicine, because of ill health, he turned over much of his practice to Baillie.

Baillie's practice grew very rapidly and at length in order to attend all his patients he found it necessary to work from 6 o'clock in the morning until 11 o'clock each night. This state of practice lasted for a period of twelve years and was ruinous to Baillie's health. Soon he was obliged to confine his work to consultation.

In 1810, Baillie received the command of King George III to attend his daughter, the Princess Amelia, who died on November 2 of the same year. This was the beginning of his many attendances to the Royal family, for he was soon appointed physician extraordinary to the King.

In the summer of 1823, Baillie's health again failed, presumably because of continued overwork. A severe cough developed, apparently the result of an inflammation of the trachea. He died on September 23, 1823. A bust of him was made by Chantrey and placed in Westminster Abbey.

Baillie made important contributions to the medical literature. In 1788 and in 1789 he published two anatomic papers in the "Philosophical Transactions" of the Royal Society. The first of these was in the form of a letter to John Hunter entitled "An Account of a Remarkable Transposition of the Viscera." This early observation of congenital dextrocardia with complete situs-transversus we are reprinting. According to Pettigrew, cases of a similar kind were recorded by Winslow, Sir Astley Cooper, Dr. Quain, Dr. Watson, and others. Baillie's second paper to the Royal Society was communicated by John Hunter. It was entitled "An Account of a Particular Change of Structure in the Human Ovarium." In 1790, presumably because of these publications, Baillie was elected to the Royal Society.

In the year 1793 he published the first edition of his "The Morbid Anatomy of Some of the Most Important Parts of the Human Body." During Baillie's lifetime this book went through several editions. In order to illustrate the subject of morbid anatomy on a systematic plan, Baillie began to publish "fasciculi" of engravings. Ten of these were published separately and in 1803 they appeared in book form. A second edition of this work appeared in 1812.

During his later years, Baillie contributed several articles to the "Transactions" of the Society for the Improvement of Medical and Chirurgical Knowledge and to the "Transactions" of the Royal College of Physicians.

In addition to membership in the Royal Society of London and the Royal Society of Edinburgh, Baillie was an honorary member of the Royal College of Physicians of Edinburgh, a Fellow of the Royal College of Physicians in London, a member of the Medico-Chirurgical Society in London, and an honorary member of the Medical Societies of Erlangen and Bonn.

OF A REMARKABLE TRANSPOSITION OF THE VISCERA*

By

MATTHEW BAILLIE

NOTHING tends more to illustrate the powers and the wisdom of nature than the investigation of the structure of animals. We there find a most wonderful delicacy of mechanism, and exquisitely adapted to a variety of purposes. This however is not to be better seen by following nature in her common track than by observing her wanderings. In these she often shows more particularly the extent of her powers, and throws light on her ordinary plans. Such circumstances give importance and value to the observation of singular phenomena. The variety in animal structure, an account of which is presented in this account, is a complete transposition in the human subject, of the thoracic and abdominal viscera, to the opposite side from what is natural. It is so extraordinary as scarcely to have been seen by any of the most celebrated anatomists, and indeed has been but very generally noticed at all. The circumstance has been mentioned, but it has not been particularly described so as to make it thoroughly known, or to establish its certainty. It was hanging in the minds of many as doubtful, whether such a variety did really exist. There is one circumstance that attends the account of the present case, which has not always happened in the record of singular phenomena, viz. that it has been examined by physicians and surgeons of the first reputation in this large town, and has been in some measure open to the gratification of public curiosity.

The person who is the subject of this paper was a male, nearly 40 years of age, somewhat above the middle stature, and of a clean active shape. He was brought for dissection in the common way to Windmill-street. On opening the cavity of the thorax and abdomen, the different situation of the viscera was so striking as immediately to excite the attention of the pupils who were engaged in dissecting it. I began immediately to examine every part of the change with considerable attention: for this purpose after desiring a drawing to be made of the appearances as they were found on opening the body, I next day injected it.

The mediastinum or anterior duplicature of the pleura, separating the 2 cavities of the chest from each other, was found to incline obliquely

*Phil. Tr. Roy. Soc. London 16: 183-189, 1785-1790 (abridged 1809). (Original 78: 350, 1788.)

downwards to the right side fully as much as it does commonly to the left side of the chest The pericardium too inclined obliquely to the right side On pressing it gently away from the lungs the phrenic nerves came distinctly into view, in their common situation, but the right phrenic nerve ran more obliquely, and was longer than the left The lung on the right side was divided by a single oblique fissure into 2 lobes, having at the same time a deficiency opposite to the apex of the heart, and the lung on the left side was divided into 3 lobes, exactly contrary to what is found in ordinary cases

On opening the pericardium the apex of the heart was found to point to the right side nearly opposite to the 6th rib, and its cavities as well as large vessels were completely transposed What are commonly called the right auricle and ventricle were situated on the left side, and the left auricle and ventricle on the right The pulmonary artery ascended towards the right side of the chest The aorta was also directing its arch to the right, and the vena cava superior, as well as inferior, were seen opening into their auricle on the left side of the spine There was nothing remarkable in the size or general figure of the heart On the outside of the pericardium the transposition of the larger vessels was very striking The longer subclavian vein was passing from the left side obliquely to the right before the branches which are sent off from the arch of the aorta The left carotid and subclavian arteries were found to arise from the arch of the aorta by one common trunk, the right carotid and subclavian separately

In the duplicature of the pleura behind, or what may be called the posterior mediastinum, there was a change corresponding to what we have already described The descending aorta was found passing on the right side of the spine The oesophagus was before it, inclining more and more to the right towards its lower extremity, and it at length perforated the diaphragm somewhat on the right side of the spine* The thoracic duct was seen in the middle between the descending aorta and vena azygos, in some places forming a plexus of small branches, in another dividing itself into 2 branches, which afterwards re-united in a common trunk, and at length climbing up to terminate in the angle between the jugular and subclavian veins on the right side of the body The recurrent nerve of the parvagum on the right side passed round the beginning of the descending aorta, and on the left passed round the common trunk of the carotid and subclavian arteries The large intercostal nerves being exactly under the same circumstances on each side, it was impossible there could be any transposition in them It appears then from the foregoing description, that every thing admitting of such a change was completely transposed in the thorax

*The vena azygos was on the left side of the spine opening in the common way into the vena cava superior, which we formerly mentioned to be also transposed in its situation —Orig

The liver was situated in the left hypochondriac region, the small lobe being towards the right, and the great lobe in the left side The ligaments uniting it to the diaphragm corresponded to this change, the right transverse ligament being longer, and the left being shorter, than usual The suspensory ligament could undergo little change, except being pushed to the left side along with the liver On pressing upwards the liver, so as to exhibit its posterior and under surface, the gall bladder was seen on the left side preserving its proper relative situation to the great lobe of the liver, and the vessels of the portae were found on dissection to be transposed corresponding to the change of circumstances The hepatic artery was found climbing up obliquely from the right towards the left, before the lobulus spigelii, and entered at the portae into the substance of the liver by two or three branches on the right of the other vessels The ductus communis cholidochus was on the left of the other vessels, being formed from the ductus hepaticus and ductus cysticus in the common way, and it passed obliquely downwards on the left, to terminate in the duodenum What was most remarkable, it terminated in the fore part of the duodenum The vena portarum passed behind the hepatic artery and ductus communis cholidochus, ascending obliquely towards the left side

The spleen was situated in the right hypochondriac region, adhering to the diaphragm in the common way There were 3 spleens, nearly of the size of a pullet's egg, found adhering to the larger spleen by short adhesions, besides 2 other still smaller spleens which were involved in the epiploon at the great end of the stomach The pancreas was found on the right side behind the stomach, running obliquely from the spleen to the curvature of the duodenum, and had its duct entering in common with the ductus communis cholidochus into the cavity of that intestine The splenic vessels were passing along the upper edge of the pancreas to the right side, corresponding to the change of situation in the pancreas and spleen

The stomach was situated on the right side, partly hid by the small lobe of the liver passing to the left, and terminating in the pylorus, rather on the left side of the spine The duodenum took a most singular course, it first passed to the right side, behind the small end of the stomach, it then turned on itself, towards the left side, it afterwards took its proper sweep to the right side, passing behind the superior mesenteric artery and mesaraica major vein The mesentery began to be formed on the right side, instead of the left, as in ordinary cases The ilium terminated in the great intestine on the left side, and there was in it a diverticulum of considerable size, a lusus not unfrequently occurring The caecum was situated on the left psoas magnus and iliacus internus muscles The transverse arch of the colon passed from the left to the right side of the body, and the sigmoid flexure crossed over the right psoas, to get into

the cavity of the pelvis The kidneys had their vessels transposed, the renal capsules had undergone no change, as no variety could be produced by a transposition

The aorta passed between the crura of the diaphragm into the cavity of the abdomen, and adhered in its course to the spine on the right side of the vena cava inferior Its branches were directed in their course corresponding to the peculiar situation of the viscera The splenic and coronary arteries were passing to the right side, and the hepatic artery obliquely to the left The superior and inferior mesenteric arteries were directed to the right side There was no change in the spermatic arteries, any transposition in the testicles, if such a thing could take place, not being capable of affecting them The lumbar arteries could also undergo little change, except that the left lumbar arteries must necessarily, from the peculiar situation of the aorta, be the longest The vena cava inferior perforated the tendinous portion of the diaphragm, and adhered in its course to the spine on the left side of the aorta

The right emulgent vein was much longer than usual, passing from the right kidney before the aorta to terminate in the vena cava superior, and the left emulgent much shorter, passing from the left kidney to the vena cava, which was situated on the left side of the spine The right spermatic vein was found to open into the right emulgent, and the left into the vena cava inferior, about an inch under the left emulgent The vena portarum was changed from its natural course, passing obliquely upwards to the left side, and its large branches, viz the vena splenica, mesaraica major and minor, were all directed towards the right side of the spine There was no change in the intercostal nerve within the cavity of the abdomen, nor does it seem to be capable of being affected by any transposition of parts We see then, that there was a complete transposition of the abdominal viscera, each of them preserving its proper relative situation to the others In the brain, organs of sense, of generation, the muscles, and blood vessels of the extremities, was found nothing remarkable

The person seems to have used his right hand in preference to his left, as is usually the case, which was readily discovered by the greater bulk and hardness of that hand, as well as the greater fleshiness of the arm It was not indeed to be expected he should be left handed The person, while alive, was not conscious of any uncommon situation of his heart, and his brother has his heart pointing to the left side as in ordinary cases Indeed, there was little reason to expect that we should meet with any thing particular in the account of his life His health could not be affected by such a change of situation in his viscera, nor could there arise from it any peculiar symptoms of disease Still less could there be any connection between such a change and his dispositions, or external actions He might have known that his heart was directed to-

wards the right side, but if we consider how little every person, especially those of the lower class, are attentive to circumstances not very palpable, it was scarcely to be expected he should know of it

Notwithstanding the general similarity of parts in the same species of animals, there is no reason why nature should not sometimes deviate from her ordinary plans Accordingly we find there is much variety in animal structure, but this does not commonly affect the animal functions Under this restriction the variety is so great in the appearances of every part of an animal, that it is almost impossible to examine any 2 animals of the same species without remarking many differences In the bony compages of an animal we find little variety in the extremities of bones where there is the apparatus of a joint, because a particular shape is best adapted to a particular kind or latitude of motion In other parts of the bones, where a difference of features is not material, there is great variety, as in the foramina, depressions, ridges, and sutures of bones The same general rule will apply to variety in muscles The principal object is a certain insertion near a joint, so as to give a determined direction of motion With respect to such insertions, there is, comparatively speaking, little variety, but there is a great difference in the bodies and connections of muscles, which have no share in the regulation of the motion

There is no part of an animal where there is a greater latitude of variety than in the distribution of blood vessels The reason of it is very obvious The only object in the distribution of blood vessels is, to carry blood to every part of the body and bring it back to the heart The parts of an animal, in order to be supported, must be visited by successive changes of fresh blood, but it surely cannot be an object of importance whether the blood passes by one route or another Hence the variety of blood vessels is extremely great Still, however, there is a method in the deviations of nature,—so that they may be marked or noted, the same varieties occurring in different animals

It cannot be at all important to the function of a viscus, whether it be in one mass, or in separate portions The structure being the same, the same action will take place Hence we often find the two kidneys joined together, forming one mass, and not unfrequently two or three spleens, besides the common one Neither can it be important whether a viscus should always be of the same shape, because its functions do not depend on shape, but on structure we find accordingly, in this particular, much variety

There are many of the viscera which are connected together in their functions or by the junction of large blood vessels, in such a way as to require nearly the same relative situation among themselves This becomes also necessary in order to preserve the general shape of the animal Accordingly we find that when any important viscus is changed in its

situation, it affects the situation of other viscera, requiring in them a similar change We saw in the person who is the subject of this paper, that a change in the situation of the heart and liver was accompanied with a change of situation in the stomach, spleen, pancreas and in short the whole abdominal viscera This, however, is a great deviation in nature, for it is nothing less than changing almost the whole vital system in an animal, and therefore, it rarely happens In such a change it does not appear that the functions can be affected, as they depend on structure and situation, which are both preserved Hence the person who is the subject of this paper arrived at the age of maturity and might have continued to live to an extreme old age The human machine might have been constructed in this way generally, and under such circumstances, what is now called the natural situation of parts would have been as singular as the present phaenomenon

There appears to be less variety in the nervous system of animals of the same species, than in most parts of the body There is scarcely any difference in the appearance of the brain, and much less in the distribution of the nerves than of the blood vessels There is also little variety in the organs of sense perhaps the mechanism in both these is nicer, so that a considerable deviation would interfere with their peculiar functions The most common great deviations which nature produces in the structure of an animal, are various kinds of monstrosity, by which the animal becomes often unfit for continuing its existence Why nature should in its greater deviations fall into a very imperfect formation, much below the standard of her common work, does not appear very obvious It seems that there might have been many varieties where the functions could have been preserved Perhaps it is with a view to check the propagation of great varieties, so as to preserve a uniformity in the same species of animals

It has been much agitated, whether monstrosities depend on the original formation, or are produced afterwards in the gradual evolution of an animal This does not appear to be a question of much importance, nor perhaps can it be absolutely determined But on the whole it is more reasonable to think that the same plan of formation is continued from the beginning, than that at any subsequent period there is a change in that plan It may be observed, that it is exactly the same creative action which produces the natural structure, or any deviation from it, for in cases of deviation the action is either carried too far, ceases too soon, or is diverted into uncommon channels This will explain the various kinds of monstrosity from redundancy, deficiency, or transposition of parts

1794

JOHN HUNTER

THE RECORD OF HIS CARDIAC HISTORY
AS DETAILED BY HIMSELF AND LATER
PUBLISHED BY HIS BROTHER-IN-LAW,
EVERARD HOME

JOHN HUNTER
From a painting by Sir Joshua Reynolds

(Courtesy Medical Classics)

JOHN HUNTER

(1728 1793)

"In the history of human progress there appears now and then a thunderbolt, and the thunderbolt of surgery is John Hunter "

—William Boyd *

"Not Oxford with its intellectual store
Of Greek and Latin, but the open space
Of the wide firmament for him, to face
Nature herself and Universal Law
He entered Truth's stout stronghold at the door
And, step by step, climbed up, with measured pace,
Until he reached a high embattled place
Where he remained and will, for evermore "

—T W Parry

JOHN HUNTER was born at Long Calderwood, in the parish of East Kilbride, Lanarkshire, Scotland, on February 13 or 14, 1728, the youngest of ten children Two of his brothers, James and William Hunter, also became physicians His elder brother, James, died at the beginning of a most promising career, and to William fell the task of aiding John in an unparalleled career in medicine

At the age of seventeen John Hunter was sent to live with his brother-in-law, a Mr Buchanan, where he developed great mechanical skill as a carpenter in Buchanan's workshop During this time his brother, William, had made a very successful start in the practice of medicine, and at the age of twenty John Hunter decided that he, too, would have a medical career

He therefore wrote his brother William in London and offered his services as an assistant in William Hunter's dissecting laboratory His offer was accepted and while in his brother's tutelage he was so successful that the next year he was to direct the dissections of the medical students

Not long after he came to London, Hunter was introduced by his brother to the famous English surgeon, William Cheselden He soon became a student of Cheselden, under whom he worked at Chelsea Hospital during the summer months of 1749 and 1750 On Cheselden's retirement in 1751, John Hunter became a surgical student at St Bartholomew's Hospital There he worked under the distinguished surgeon, Percival Pott

In 1753 Hunter entered St Mary's Hall, Oxford, as a student, at the suggestion, no doubt, of his brother, William There he began to study the classics, but after a trial of two months dropped his course and determined to remain within the realm of surgery In 1754 he became a surgical student at St George's Hospital Two years later he served there as house-surgeon

In 1754, Hunter after making several dissections discovered the method of connection between the placenta and the uterus This discovery twenty-five years later (1780) was to provoke a bitter public quarrel between the two brothers over the question of priority, and it is to be regretted that it was to dissolve their friendship Both of the Hunters disputed with the Monros over priority in anatomic discoveries and later with Pott on the true nature of congenital hernia

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* *Surgical Pathology* ed 4 1938 p 17

In 1759 Hunter, having served a period of ten years in the study of human anatomy, in order to understand more clearly the human body and its functions, began the study of comparative anatomy Because he worked very hard his health suffered and he was advised to seek the curative effects of a warmer climate On this account he applied for an appointment with the army and was immediately made a staff surgeon

During the Seven Years' War he accompanied the British expedition to Belle Isle in 1761 During that siege he found ample opportunity to treat gunshot wounds, a procedure for which he was later to become renowned While he was thus engaged he found time to conduct investigations in experimental physiology He studied the digestion in lizards and snakes during hibernation and among other things, the faculty of hearing in fishes

By 1763 the war was terminated and Hunter returned to London In order to increase his income he decided to teach anatomy in private classes He conducted classes in anatomy for several years During his spare time he continued his anatomic and physiologic investigations To obtain specimens for this research he obtained the bodies of animals that had died in the zoos of London and elsewhere He also purchased rare animals, when such a course was possible, spending money he could ill afford

In 1767 Hunter was elected a fellow of the Royal Society His election probably was the result of recognition of his devout interest in comparative physiology, an interest that had not been shown by the publication of original investigations, for he had not as yet begun his publications Scientists in general in London already knew of his rapidly growing anatomic museum which, one day, was to become of national significance

Hunter was appointed surgeon to St George's Hospital in 1768, and soon afterwards he was chosen a member of the Corporation of Surgeons Thereafter he obtained pupils on more advantageous terms Among the most renowned of his students, according to Garrison, were Jenner, Astley Cooper, Abernathy, Cline, Clift, Parkinson, Blizard, Home, Alanson, Wright Post, and Physick

In 1771, Hunter published the first part of his medical classic, "The Natural History of the Human Teeth" This was followed in 1778 by the second part, "A Practical Treatise on the Diseases of the Teeth" According to Garrison, Hunter was the first to study the teeth in a scientific manner and the first to recommend complete removal of the pulp before filling them

Hunter's marriage to Miss Home, sister of Sir Everard Home, took place in July, 1771 They had four children, two of whom died when very young In 1772, Hunter, being persuaded by Sir John Pringle, communicated his first paper to the "Philosophical Transactions" of the Royal Society The work concerned the digestion of the stomach after death In the fall of this year Sir Everard Home, his brother-in-law, became his student and afterward was to act as his assistant

In 1776 Hunter was appointed surgeon-extraordinary to the King That same year he was invited to deliver the Croonian Lectures before the Royal Society For the subject of these lectures he chose "Muscular Motion" In 1778 he was appointed surgeon to the new naval hospital at Plymouth The Royal Society of Belles-Lettres of Gothenburg elected Hunter a fellow in 1781, and in 1783 he was elected to membership in the Royal Society of Medicine and the Royal Academy of Surgery of Paris

Fordyce, Hunter, and others were instrumental in founding in 1783 the Society for the Improvement of Medical and Chirurgical Knowledge Although this society had but a brief life, many valuable papers are included in its "Transactions"

In 1783, Hunter procured at a cost of 500 pounds the ''kidnapped'' body of the famous Irish giant, Byrne or Obrien He disarticulated the skeleton and it occupied a prominent place in his Museum of Natural History In the same year the Royal Society conferred upon him the Copley medal in recognition of his important discoveries in natural history

In 1785, Hunter began to suffer from recurring attacks of angina pectoris He, therefore, spent a holiday at Bath and during his convalescence entrusted his medical practice to his brother-in-law, for whose ability he had a high regard In December of that year Hunter, feeling somewhat refreshed from his stay at Bath, established his famous operation for the cure of aneurysm This consisted in tying the artery at a distance high in the healthy tissues by a single ligature

Hunter was appointed deputy surgeon-general to the army in 1786 That same year he published his work on venereal disease[1] Believing that syphilis and gonorrhea were identical he inoculated himself with spirochetes and subsequently thought he observed the symptoms of both diseases This confirmed him in his belief that both diseases were identical This erroneous conception was later demolished by Philippe Ricord (1800-1889) who proved the autonomy of these diseases (1831-1837) Hunter did, however, correctly differentiate between hard chancre and chancroid ulcer

In 1786 Hunter also published his important work, ''Observations in Certain Parts of the Animal Oeconomy,'' which consisted of a revision and enlargement of many of his papers originally published in the ''Philosophical Transactions'' of the Royal Society

In 1792, Hunter transferred his surgical lectures to his brother-in-law, Home, and devoted much of his time to the completion of his famous work, ''A Treatise on the Blood, Inflammation and Gunshot Wounds'' This was not published until 1794, about a year after Hunter's death From Home's account of the life of Hunter, which prefixes this work, we are reproducing Hunter's classic description of his own fatal illness, angina pectoris, as detailed to his brother-in-law

Hunter had said that his ''life was in the hands of any rascal who chose to annoy and tease him'' And there can be no doubt but that the violent disagreements he had with his colleagues at St George's Hospital hastened his death, which occurred on October 16, 1793, following a meeting of the board of governors of St George's Hospital, at which a colleague had directed some disparaging remarks to him

The great monument to the fame of John Hunter is the Hunterian Museum of 13,000 specimens purchased by Parliament some time after his death for 15,000 pounds, a fraction of its cost, and presented to the Corporation of Surgeons, soon afterward to become the Royal College of Surgeons

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[1]Hunter, John *A treatise on the venereal disease*, London 1786 398 pp

A

TREATISE

ON

THE BLOOD,

INFLAMMATION,

AND

GUN-SHOT WOUNDS,

BY THE LATE

JOHN HUNTER.

TO WHICH IS PREFIXED

A SHORT ACCOUNT OF THE AUTHOR's LIFE,

BY HIS BROTHER-IN-LAW,

EVERARD HOME

IN TWO VOLUMES, FROM THE LONDON QUARTO.

VOL. I

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A TREATISE ON THE BLOOD, INFLAMMATION, AND GUN-SHOT WOUNDS*

BY THE LATE JOHN HUNTER

A Short Account of the Author's Life†
By His Brother-in-Law
Everard Home

MR HUNTER was a very healthy man for the first forty years of his life, and, if we except an inflammation of his lungs in the year 1759, occasioned most probably by his attention to anatomical pursuits, he had no complaint of any consequence during that period In the spring of 1769, in his forty-first year, he had a regular fit of the gout, which returned the three following springs, but not the fourth, and in the spring of 1773, having met with something which very forcibly affected his mind, he was attacked at ten o'clock in the forenoon with a pain in the stomach, about the pylorus, it was the sensation peculiar to those parts, and became so violent that he tried change of position to procure ease, he sat down, then walked, laid himself down on the carpet, then upon chairs, but could find no relief, he took a spoonful of tincture of rhubarb, with thirty drops of laudanum, without the smallest benefit While he was walking about the room he cast his eyes on the looking glass and observed his countenance to be pale, his lips white, giving the appearance of a dead man This alarmed him, and led him to feel for his pulse, but he found none in either arm, he now thought his complaint serious, several physicians of his acquaintance were then sent for, Dr William Hunter, Sir George Baker, Dr Huck Saunders, and Sir William Fordyce, all came but could find no pulse, the pain still continued, and he found himself at times not breathing Being afraid of death soon taking place if he did not breathe, he produced the voluntary act of breathing, by working his lungs by the power of the will the sensitive principle, with all its effects on the machine not being in the least affected by the complaint In this state he continued for three-quarters of an hour, in which time frequent attempts were made to feel the pulse, but in vain, however at last, the pain lessened and the pulse returned although at first but faintly, and the involuntary breathing began to take place, while in this state he took Madeira brandy, ginger etc but did not be-

*The first English edition was published in 1794 We are reprinting from the first American edition published in 1796—F A W 1940

†Pp xxix-liii

lieve them of any service, as the return of health was very gradual, in two hours he was perfectly recovered

In this attack there was a suspension of the most material involuntary actions, even involuntary breathing was stopped, while sensation with its consequences, as thinking and acting with the will, were perfect, and all the voluntary actions were as strong as before

Quere What would have been the consequence of his not having breathed by means of the voluntary muscles? It struck him at the time that he would have died, but we cannot suppose that would have been the consequence, as breathing most probably is only necessary for the blood while circulating, and as the circulation was stopped, no good could have arisen from breathing

When he was at first attacked, the pulse was full and eight ounces of blood were taken away, but this did not appear to be of service, the day following he was cupped between the shoulders and had a large blister applied upon the part, he took an emetic, and several times purging medicines and bathed his feet in warm water, but nothing appeared to be of the least use The purging and vomiting distressed him greatly, for both the stomach and intestines were so irritable that less than half the usual quantity had the desired effect He took some James's powder,* and drank some white wine whey on account of the heat in the skin, especially in the feet and hands, which took it off and gave him for the first time a comfortable feel At the end of ten days all his ideas of his present state became more natural, the strange deception concerning his own size was in part corrected, and the idea of suspension in the air became less, but for some time after, the fire appeared of a deep purple red When he got so well as to be able to stand without being giddy, he was unable to walk without support, for his own feelings did not give him information respecting his centre of gravity, so that he was unable to ballance his body, and prevent himself from falling

He gradually recovered from this state, and as soon as he was able went to Bath, where he staid some time and drank the waters, which were thought to be of service to him, but did not stay long enough to give them a fair trial, he returned to town much better, and in a few weeks got quite well From this period to 1785, he had no particular indisposition, but certainly did not enjoy perfect health, for in 1785, he appeared much altered in his looks, and gave the idea of having grown much older than could be accounted for from the number of years which had elapsed

About the beginning of April, 1785, he was attacked with a spasmodic complaint, which at first was slight, but became afterwards very violent,

*James's powder contained 1 part of antimonious oxide and 2 parts of calcium phosphate and was used as a diaphoretic —F A W 1940

and terminated in a fit of the gout in the ball of the great toe, this, like his other attacks, was brought on by anxiety of mind, the first symptom was a sensation of the muscles of the nose being in action, but whether they really were or not, he was never able to determine, this sensation returned at intervals for about a fortnight, attended with an unpleasant sensation in the left side of the face, lower jaw, and throat which seemed to extend into the head on that side and down the left arm, as low as the ball of the thumb, where it terminated all at once, these sensations were not constant, but returned at irregular times, they became soon more violent, attacking the head, face, and both sides of the lower jaw, giving the idea that the face was swelled, particularly the cheeks, and sometimes slightly affected the right arm After they had continued for a fortnight they extended to the sternum, producing the same disagreeable sensations there, and giving the feel of the sternum being drawn backwards toward the spine

I was with him during the whole of this attack, and never saw anything equal to the agonies he suffered, and when he fainted away, I thought him dead, as the pain did not seem to abate, but to carry him off, having first completely exhausted him

He then fell asleep for half an hour, and awoke with a confusion in his head, and a faint recollection of something like a delirium, this went off in a few days

The affections above-described were, in the beginning, readily brought on by exercise, and he even conceived that if he had continued at rest, they would not have come on, but they at last seized him when lying in bed, and in his sleep, so as to awaken him, affections of the mind also brought them on, but coolly thinking or reasoning did not appear to have that effect While these complaints were upon him, his face was pale, and had a contracted appearance, making him look thinner than ordinary, and after they went off his colour returned, and his face recovered its natural appearance On the commencement of the complaint, he suspected it to be rheumatism, and applied electricity to his arm, which took it off for the time only, he then, for two or three nights successively, took three grains of James's powder, without any abatement of the symptoms, he next had recourse to the camphorated julep, both at the commencement of the spasm, and while it was upon him, but obtained no relief, he tried Hoffman's anodyne liquor, in the dose of a tea-spoonful, and not finding it to answer alone, joined to it the camphorated julep, but the spasms seemed to be more violent, one night he took twenty drops of thebaic tincture, which made his head confused all the following day, but did not at all abate the spasms, the following day he took two tea-spoonfuls of the bark, which heated him, and gave him a head-ach, thirst, and dryness of his mouth, which prevented his continuing it At the desire of Dr David Pitcairn,

he took the powder of valerian, an ounce a day, which seemed for the first two days to remove his spasms, but they returned on the third with more violence than usual, especially one evening at the Royal Society, which induced him to leave off the valerian, and he bathed his feet on going to bed in warm water, mixed with half a pound of flour of mustard, and took a tea-spoonful of tincture of rhubarb in ginger-tea, also wore worsted stockings all night

On Friday morning, the twentieth of May, between six and seven o'clock, he had a violent spasm, attended with most violent eructations of wind from the stomach for nearly a quarter of an hour Dr Pitcairn, who was sent for upon this occasion, asked him, if there was any distress upon his mind that had brought on this attack, and he confessed his mind to have been much harrassed, in consequence of having opened the body of a person who died from the bite of a mad dog, about six weeks before, in doing which he had wounded his hand, and for the last fortnight his mind had been in continual suspense, conceiving it possible that he might be seized with symptoms of hydrophobia This anxiety preying upon his mind for so long a time, there is every reason to believe was the cause of the present attack and probably had also brought on the former ones, which were all after the accident which had impressed his mind with this horrible idea

At the desire of Dr Pitcairn, he took at two doses in the forenoon, ten grains of asafoetida, and three grains of opium, and in the afternoon fifteen of asafoetida, and one of opium, in the evening he had a head-ach, which was supposed to be brought on by the opium, his bowels were loaded and oppressed with wind, and he endeavoured in vain to procure a motion by laxative clysters, although repeated, and ten grains of jalap were taken by the mouth, he passed a very restless night On Saturday morning he was visited by Sir George Baker, Dr Warren, and the late Dr Pitcairn he repeated the asafoetida twice in the course of the day, and two spoonfuls of the following mixture were taken every hour, without producing a motion till about half an hour after the whole was used

Infusion of senna, six ounces,
Tincture of senna, one drachm and a half,
Soluble tartar, three drachms

M

In the afternoon he had another evacuation, soon after which, the most violent attack of spasm which he had experienced, came on, nothing was attempted internally during the attack, which lasted two hours, a bladder of hot water was applied to the heart, and afterwards to the feet, without any effect

The asafoetida was now left off, and this evening he began the oleum succini in saline draughts fifteen drops every six hours On Sunday morning he continued the oleum succini, but the saline draught was changed to cinnamon-water, and a large blister was put upon the back

close to the neck, he continued pretty free from spasm On Monday the blister was taken off, and the oleum succini continued, but about nine o'clock at night he had threatenings of spasm, with head-ach, and the feel of a load in his bowels, he had a pain in the left side and region of the stomach, with violent eructations of wind from the stomach, which lasted about two hours, he took thebaic tincture, twenty-five drops, in the warm tincture of rhubarb, and afterwards some baume de vie, but the eructations continuing, sinapisms were applied to the feet, after which they ceased, and the sinapisms were so troublesome that he had them taken off five hours after they were applied On Tuesday morning he felt himself easier, the oleum succini was continued, five drops of laudanum being added to each dose, in the evening he bathed his feet in warm water, to clean them from the sinapisms, and both the great toes appeared a little inflamed, and very tender, they were more painful after being bathed, and were very troublesome all night On Wednesday morning the inflammation and swelling in the great toes appeared evidently to be the gout, and the pain continued very acute till Thursday, when it began to abate and on Friday was very much diminished he continued the oleum succini on Wednesday, and took a bolus of aromatic species before each dose, but on Friday the oleum succini made him sick, and was left off On Saturday he began the bark in tincture and decoction with the species aromaticae, Sunday continued the bark, and having eructations and flatulencies after his meals, he was ordered every day before dinner, rhubarb fifteen grains, ginger ten grains, in a bolus He had no spasm after Monday the thirtieth of May, he however had threatenings, or slight sensations, similar to those which preceded the spasms, and occasional eructations Although evidently relieved from the violent attacks of spasm by the gout in his feet, yet he was far from being free from the disease, for he was still subject to the spasms, upon exercise or agitation of mind, the exercise that generally brought it on, was walking, especially on an ascent, either of stairs or rising ground, but never on going down either the one or the other, the affections of the mind that brought it on were principally anxiety or anger it was not the cause of the anxiety, but the quantity that most affected him, the anxiety about the hiving of a swarm of bees brought it on, the anxiety lest an animal should make its escape before he could get a gun to shoot it, brought it on, even the hearing of a story in which the mind became so much engaged as to be interested in the event, although the particulars were of no consequence to him, would bring it on, anger brought on the same complaint and he could conceive it possible for that passion to be carried so far as totally to deprive him of life but what was very extraordinary the more tender passions of the mind did not produce it, he could relate a story which called up all the finer feelings as compassion admiration for the actions of gratitude in others so as to make

him shed tears, yet the spasm was not excited, it is extraordinary that he eat and slept as well as ever, and his mind was in no degree depressed, the want of exercise made him grow unusually fat

In the autumn 1790, and in the spring and autumn 1791, he had more severe attacks than during the other periods of the year, but of not more than a few hours duration in the beginning of October, 1792, one, at which I was present, was so violent that I thought he would have died On October the 16th, 1793, when in his usual state of health, he went to St George's Hospital, and meeting with some things which irritated his mind, and not being perfectly master of the circumstances, he withheld his sentiments, in which state of restraint he went into the next room, and turning round to Dr Robertson, one of the physicians of the hospital, he gave a deep groan, and dropt down dead

At the time of his death he was in the 65th year of his age, the same age at which his brother, the late Dr Hunter, died Upon inspecting the body after death, the following were the appearances the skin in several places was mottled, particularly on the sides and neck, which arose from the blood not having been completely coagulated, but remaining nearly fluid

The pericardium was very unusually thickened, which did not allow it to collapse upon being opened, the quantity of water contained in it was scarcely more than is frequently met with, although it might probably exceed that which occurs in the most healthy state of these parts The heart itself was very small, appearing too little for the cavity in which it lay, and did not give the idea of its being the effect of an unusual degree of contraction, but more of its having shrunk in its size Upon the under surface of the left auricle and ventricle, there were two spaces nearly an inch and an half square, which were of a white colour, with an opaque appearance, and entirely distinct from the general surface of the heart these two spaces were covered by an exudation of coagulating lymph, which at some former period had been the result of inflammation there The muscular structure of the heart was paler and looser in its texture than the other muscles in the body There were no coagula in any of its cavities The coronary arteries had their branches which ramify through the substance of the heart in the state of bony tubes, which were with difficulty divided by the knife, and their transverse sections did not collapse, but remained open The valvulae mitrales, where they come off from the lower edge of the auricle, were in many places ossified, forming an imperfectly bony margin of different thicknesses, and in one spot so thick as to form a knob, but these ossifications were not continued down upon the valve towards the chordae tendineae

The semilunar valves of the aorta had lost their natural pliancy, the previous stage to becoming bone, and in several spots there were evident ossifications

The aorta immediately beyond the semilunar valves had its cavity larger than usual, putting on the appearance of an incipient aneurism, this unusual dilatation extended for some way along the ascending aorta, but did not reach so far as the common trunk of the axillary and carotid artery The increase of capacity of the artery might be about one-third of its natural area, and the internal membrane of this part had lost entirely the natural polish, and was studded over with opaque white spots raised higher than the general surface

From this account of the appearances observed after death, it is reasonable to attribute the principal symptoms of the disease to an organic affection of the heart That organ was rendered unable to carry on its functions, whenever the actions were disturbed, either in consequence of bodily exertion, or affections of the mind

The stoppage of the pulse arose from a spasm upon the heart, and in this state the nerves were probably pressed against the ossified arteries, which may account for the excruciating pain he felt at those times

The other symptoms may be explained from the defect in the valves and the dilatation of the aorta, which had lost its elasticity

In the last attack the spasm upon the heart was either too violent in the degree of contraction, or too long continued to admit of relaxation, so that death immediately ensued

His remains were interred in a vault under the parish church of St Martin in the Fields, attended by a few of his oldest medical friends

1806

JEAN NICOLAS CORVISART

DESCRIPTION OF THE SIGNS OF CONTRACTION OF THE ORIFICES OF THE HEART, ETC.

JEAN NICOLAS CORVISART

Portrait by Charles Bazin, etched by Delpech

(Courtesy Charles C Thomas)

JEAN NICOLAS CORVISART

(1755-1821)

IN THE tiny French village of Dricourt, not far from Vouziers, Jean Nicolas Corvisart was born on February 15, 1755. As was to happen later to Laennec, Corvisart early in life was sent to live with his uncle, who was the pastor of the parish at Vimille.

At the age of thirteen, Corvisart matriculated at the College of Saint Barbe. There he showed no signs of intellectual superiority, but, according to Beeson, was "lazy, mischievous, and quarrelsome." Originally it was intended that Corvisart was to become a lawyer, but while studying for the bar, he visited many hospital clinics in Paris. These visits made a profound impression on him, so he decided to forsake law for medicine.

This decision angered his father to such an extent that Corvisart was expelled from the paternal home. He immediately applied at the Hôtel-Dieu in Paris for a position which would insure his board and room. This he secured and with it came the splendid opportunity of studying medicine in one of the world's most famous institutions.

In 1785, the Paris Faculty of Medicine conferred upon him the degree of doctor-regent. Corvisart later became an associate to the chair of anatomy in the Hôtel-Dieu. He also assisted in the courses in operative surgery, obstetrics, and physiology.

On the death of Desbois de Rochefort in 1788, Corvisart succeeded him as physician to the Charité Hospital, and with the establishment of the medical school in 1795, Corvisart was chosen to occupy the chair of medicine. In 1797 he became professor of medicine at the Collège de France, and in 1799, Corvisart and Barthez became physicians to the government.

In 1804, Corvisart had the honor to become personal physician to Napoléon Bonaparte. He had known Napoléon at an earlier date, for in 1798 Bonaparte had presented him with a large carved mahogany chair which Corvisart had placed in his consulting rooms. This chair, now a highly valued antique, is still a cherished possession of the Corvisart family.

From all accounts, Napoléon and Corvisart greatly enjoyed each other's company, and many interesting anecdotes are told of their conversations. In one of these, it is said that the Emperor, who was in constant fear of being poisoned, literally rolled on the floor because of a slight attack of indigestion. Corvisart is supposed to have reproved him, saying, "Get up! What would be said if the master of the world were seen thus crushed by fear?"

Perhaps the most famous story is the reported conversation related by Beeson, between Corvisart and the Emperor following the birth of Napoléon's son, the King of Rome, in 1811.

"Sire," said the physician, "this child should fulfill your last wish. Consider from what a position you have arisen in less than ten years: lieutenant, captain, brigadier-general, general-in-chief, First Consul, Emperor, spouse of an Austrian Archduchess, and the father of a male child. You have reached the summit of the Wheel of Fortune and of great renown. Stop! Sire, or Destiny may desert you and then nothing remains but downfall and disaster."

''Well,'' replied the Emperor, ''that was such a speech as one would expect from a native of the Champagne ''

In reading the works of the famous Viennese physician, Max Stoll, Corvisart learned of Auenbrugger's treatise on percussion He became very much interested in this new diagnostic procedure, and after practicing it for several years, translated Auenbrugger's treatise in 1808, adding several original observations As we have mentioned in our biographic account of Auenbrugger, much credit is due to Corvisart for bringing into professional favor the discovery and use of percussion as an important aid in physical diagnosis

Corvisart realized the importance of presenting to French medicine in translation the works of some of the important foreign physicians In 1797 he translated Max Stoll's masterful work ''Aphorisms on the Knowledge and Cure of the Fevers '' He also translated the aphorisms of the great Boerhaave in 1802 In this connection it is interesting to note that many years later a favorite pupil, Laennec, published the aphorisms of his learned teacher, Corvisart

The many contributions of Corvisart to cardiology are contained in his work entitled ''Essai sur les maladies du coeur et des gros vaisseaux,'' which was first published in 1806 Among the outstanding contributions to this field were his observations on the distinctions between cardiac and pulmonary disorders and the differences between functional and organic cardiac disease Beeson wrote that Lassus was ''of the opinion that Corvisart's most important contributions to cardiology were (1) the distinction between cardiac hypertrophy and dilatation, (2) the division of the clinical course of heart failure into three periods, and (3) the connection between cause and effect in valvular lesions and heart failure '' We have chosen to reproduce from Gate's translation of the ''Essai,'' published in 1812, some of Corvisart's original observations

During the peak of his career, Corvisart was physician to many distinguished women of the time, including the Empress Josephine, the Empress Marie Louise, Hortense de Beauharnais, who was Queen of Holland, the Queen of Spain, the beautiful Pauline Bonaparte, who like her mighty brother died of cancer, and Madame Walewska, whom he is said to have dearly loved

In the short interlude of Napoléon's return to power before his defeat at Waterloo, Corvisart again served as physician to his old friend Soon after the Emperor's final defeat, Corvisart retired, it is said, to Courbevoie near Paris In 1815 he suffered a mild cerebrovascular seizure, following which he retired from the practice of medicine On September 15, 1821, he suffered a final attack, which resulted in his death a few days later, approximately four months after the demise of the former Emperor at St Helena on May 5

AN

ESSAY

ON THE

ORGANIC DISEASES AND LESIONS

OF THE

HEART AND GREAT VESSELS.

FROM THE CLINICAL LECTURES OF

J. N. CORVISART,

First Physician of their Imperial and Royal Majesties, Officer of the Legion of Honor, Honorary Professor of the School of Medicine of Paris, and of the Imperial College of France. Physician in Chief of the Hospital of La Charité, &c. &c.

PUBLISHED, UNDER HIS INSPECTION, BY

C. E. HOREAU,

Doctor in Medicine, Surgeon of the Infirmary and House of the Emperor and King.

Hæret lateri lethalis arundo.
VIRG. ÆNEID.

TRANSLATED FROM THE FRENCH,

WITH NOTES,

BY JACOB GATES, M. M. S. S.

BOSTON:

PUBLISHED BY BRADFORD & READ, AND BY

ANTHONY FINLEY, PHILADELPHIA.

1812.

ESSAY ON THE DISEASES AND ORGANIC LESIONS OF THE HEART*

THIRD CLASS, CHAPTER III, ARTICLE III

OF THE SIGNS PECULIAR TO THE CONSTRICTIONS OF THE ORIFICE†

THE cartilaginous or osseous induration of the orifices of the auricles and ventricles, of the mitral and tricuspid valves, of the aortic and pulmonary semi-lunar valves, and the vegetations, growing upon either the ventricular or arterial valves, tend principally to produce a more or less complete constriction of the orifices affected

When these constrictions exist, the circulation is embarrassed, and its phenomena singularly perverted By observing the disorder of the circulation, the practitioner may find, in the living man, I should presume, certain signs of this species of affection

To point out with accuracy these signs, it is necessary to make a distinction between the different affections of which I have been speaking 1st, those which produce a permanent obliteration of the orifices, 2d, those which form this constriction but momentarily

In the first rank, must be placed the indurations, and ossifications of the circles and ventricular valves, as the effects of this permanent morbid state are the same in every instance, and are ever perceptible to the practitioner In the second, must be placed the vegetations, or excrescences which are noticed upon the ventricular and sigmoid valves, whose presence is known only at intervals, when these bodies, generally floating in the cavity of the ventricle, or vessel, appear at the orifice, and edge where their base is fixed

The signs of constriction are commonly the more obscure, as the constriction is the more remote from the beginning of the general arterial system, because it is by considering attentively the derangements which obtain in the action of this system, that the knowledge of the signs is acquired I will explain myself the constrictions which are formed by the ossifications surrounding the orifices or valves of the right heart, as well as of the pulmonary artery, appear very difficult to comprehend on the living sub-

*Corvisart, Jean Nicolas *Essai sur les maladies et les lésions organiques du coeur,* Paris 1806 English translation by Jacob Gates M M S S 1812 pp 182-189 and 299-303

†M Corvisart planned his book on the basis of the medico-anatomic aspects of the heart He used the word class instead of the usual "part' or 'section to indicate various portions of his work beginning with 'First Class and ending with 'Fifth Class and "Corollaries" The portions reprinted herein are from the 'Third Class' and the "Corollaries,' respectively—F A W, 1940

ject Where can we, in short, discover the signs proper to make them understood? The regular or perverted action of the right cavities of the heart are barely made sensible in the organs subordinate to the influence of the less circulation, likewise the disorder of the action of the left heart can be comprehended only in the nature of the arterial pulsation, or what amounts to the same, in the phenomena of the greater circulation

Could we examine the pulsations of the pulmonary artery or its branches, as we do the pulsations of the aorta or its branches, we should recognize with equal ease, both the constrictions of the orifices of the right heart, and the same lesions when they obtain in the orifices of the left cavities, but such an investigation is impossible, and we are obliged, in this case, to examine the state of the lungs Now, is it known, or shall we be ever able to know what disorder, or rather what modification respiration will undergo, when the lungs, from a constriction either of the ventricular orifice of the right side, or of the mouth of the pulmonary artery, will receive a less quantity of blood than what ought naturally to be conveyed to them, and upon which respiration exercises its vivifying influence? It is supposed that a particular modification of respiration must furnish the signs which I am endeavoring to discover, but such a modification is not sufficiently prominent, and striking, or rather we are not endowed with sufficient understanding, to recognize such a peculiar modification of respiration among the multitude of signs which this function evinces in the different affections of the lungs

If we cannot comprehend the signs of the constrictions of the right orifices of the heart, can we expect to be more successful in a particular change of the phenomena of the greater circulation? The influence of one of the two circulations over the other is such, that, the one being disordered, it is impossible for the other not to partake of the disorder But what can be the peculiar nature of the disorder which must exist? I doubt whether observation gives the practitioner a diagnosis subtile enough to discriminate the alteration of these orifices in the particular disorder of respiration, unless he be assisted by all the concomitant signs

By reasoning physiologically, it may be advanced that the small quantity of blood conveyed from the right cavities of the heart, into the lungs, thence into the cavities of the left heart, and filling these cavities partially, will stimulate them imperfectly, that from this insufficient stimulus will arise feeble and slow contractions, which will induce debility, softness, and slowness of the pulse, etc But in this case, as in various others, to how many gross mistakes, would not he be incessantly exposed who should so restrict the morbid phenomena to the opinions of physiology, and who should always find in these too often hypothetical opinions, the knowledge of the phenomena which are to characterize any such affection! How often does not clinical observation overturn such theoretical specula-

tions, as it will subvert sooner or later many others, whose foundations appear as unstable as those too frequently established by the spirit of innovation!

Therefore the collection of a great number of symptoms would be necessary to elucidate the diagnosis of the constrictions of the right orifices, the countenance must appear of a livid hue, a more marked engorgement of the general venous system, and particularly of the liver, the size of this organ increased, the dyspnoea greater and longer continued, all the signs, in fact, which can point out the affection of the right cavities, which are usually dilated in consequence of constrictions of the right orifices, are joined to the character of the pulse, which, in this case, is less irregular than in the constrictions of the left orifices, but less regular however than natural.

The obscurity, involving the signs of the constrictions of the right orifices, is not entirely dissipated, when it is requisite to recognize the imperfect obliteration of the left auricle and ventricle. Beside the general signs of the diseases of the heart, which are ever found in this case as in the first, because an aneurismal complication generally obtains, some particular signs manifest the affections in question.

Of the preceding number of symptoms is a peculiar rushing like water, difficult to be described, sensible to the hand applied over the precordial region, a rushing which proceeds, apparently, from the embarrassment which the blood undergoes in passing through an opening which is no longer proportioned to the quantity of fluid which it ought to discharge. The same rushing is also recognized, though it is much less marked, by the hand that investigates the phenomena of the pulse. This character is not likewise the only one by which the pulse announces the existence of the contraction of the left orifice, it is effectually less regular than in the case of constriction of the right orifices, but less irregular than when the aortic orifice is deranged. Besides it presents neither impetus, hardness, nor fulness, because the quantity of blood which the left ventricle propels, is proportioned to what it receives from the auricle which is imperfectly emptied, moreover, the action of this ventricle cannot be vigorous, since it is feebly stimulated.

Notwithstanding the deficient excitement of the left ventricle, it must not be believed that in this case, the strokes and palpitations of the heart are continually weak and obscure. The right cavities, and the left auricle in particular, acquire very often a thickness and force which render the contractions extremely distinct, they may even become more violent in this case than in any other, as the strokes of the heart depend on the motion of the auricles which propels this organ, and the force of this motion of the auricles augments in proportion to the difficulty they encounter in driving the blood through their constricted orifice.

When the indurated and ossified semi-lunar valves of the aorta stop a portion of this vessel, the obstacle which they form breaks the wave of blood propelled by the heart into the artery, strong and frequent palpitations supervene, because the heart is easily filled, but is difficultly emptied, thence results a more protracted residence of the blood in the left cavities, a longer application of the stimulus of the blood on the parietes of the heart, in fact, a greater irritation of the organ The pulse, in this case, may preserve a certain degree of hardness, and rigidity, but never much fulness or regularity This continual irregularity, often increased by the frequency and force of the palpitations will always be sufficient to establish the precise diagnosis of the constriction of the aortic mouth, or the lesion of its valves Here is no obscurity, the physician with practice and attention ought ever to pronounce with confidence, and his diagnosis can no longer be uncertain though he should have for a guide only this species of undulation, this rushing noise, dull trembling, the characters so manifest by the pulse in every case of this nature

Case XL A coachman, forty-eight years of age, robust, and of a sanguine temperament, had, three days before his admission into the hospital, suffered a very severe peripneumony, treated mostly by venesection He was hardly convalescent, when he came to the hospital, May 28, 1800 I requested him to go into the Clinical Hall for the purpose of examining his pulse, which indicated an organic lesion of the heart

The pulse was very full, and even rigid, on the right side, small, soft, obscure and scarcely sensible on the left, but irregular, *undulating* and *tremulous* on each side

He was attacked with haemoptysis and very considerable suffocation, the suffocation appeared instant, the eyes were wild, the face injected, the thorax here and there painful did not sound toward the region of the heart and the lower part of the right side The pulse preserved the same character

From the effects of percussion, difficulty of breathing, haemoptysis, and characters of the pulse, I recognized the existence of hydrothorax which I judged to be subsequent to an organic lesion of the heart with constriction of the aortic orifice

The patient, while he remained here, was bled several times, and obviously relieved, but greatly debilitated, an obstinate costiveness required the use of mild purgatives

Sometime after the infiltration, which already existed, increased, diuretics, aperients, and anti-spasmodics, procured but very little relief The patient had no rest, and was obliged to sit up night and day, when the hand was applied over the region of the heart, he was endangered with suffocation He became a victim to this series of symptoms, 5th

June, eight days after his admission into the hospital, and four after the peripneumony with which he was affected

On opening the body, there was much water in the right cavity of the thorax, but little in the left The lungs were hard and adhering to the pleura The pericardium contained no serum, the size of the heart was much enlarged, the right auricle and ventricle presented nothing remarkable The left auricle was sound, with a large orifice, upon the valves of which was observed an incipient ossification The left ventricle was hard, thick, and very fleshy, the tendons of the valves were nearly ossified The aortic valves were ossified and united so closely that the end of the little finger could scarcely be introduced into the orifice of the aorta, this artery was dilated, rugous and thickened to the end of its curvature The left subclavian artery was about an inch from its origin so constricted as scarcely to admit the head of a large pin The constriction was owing to the osseous thickening of the arterial parietes

The nearly complete obliteration of the left subclavian artery explains perfectly why the pulse was scarcely sensible on this side, but this singular character could not obscure the diagnosis sufficiently demonstrated by the other symptoms, and particularly by the continual irregularity of the pulse

To conclude what I have to say on the signs of the constrictions, it remains for me to speak of the cases where the obliteration of the orifice is momentarily produced by the presence of an excrescence, vegetation, or polypous concretion on this part

When these vegetations are found appended to the mitral valves, they are announced by all the signs peculiar to the constriction of the left auricular orifice, with this exception, that most of these signs are reproduced, in this last case, only at intervals more or less remote Though, during the contractions of the left ventricle, these appendages, constantly floating at the aortic mouth, continue applied against the walls of the vessel, their effects are then very nearly the same as those produced by the simple ossification of the semi-lunar valves of the aorta, and the constriction which this ossification occasions in the aperature of the vessel, but do these excrescences, by their weight, or any other cause, compress the valves, and appear at the aortic orifice, during the contraction even of the ventricle, it is obvious that a momentary and nearly total obliteration of the aperture of the aorta will follow This closure intercepts imperfectly, for some instants, the passage of the blood, thence the successive regularities and irregularities of the pulse, the frequent and partial syncopes, this momentary interruption of the circulation compels the heart to redouble its efforts to surmount the obstacle that opposes it, thence the repeated strokes and violent palpitations of this organ which cease as soon as the obstacle is removed, and which are renewed when it is reproduced, thence the impossibility, sometimes very

protracted, of feeling the pulse, while within an instant, these pulsations return with momentary force, vivacity, frequency and regularity

I have nothing to say of the signs of the pulmonary sigmoid and tricuspid valves, as I have but seldom had the opportunity of observing them, besides, I think, as it has been already remarked, that, though this morbid state should be more frequent, it would be as difficult to indicate the particular signs of the kind of affection, as it is to distinguish the constrictions of these same orifices from those of the left cavities

COROLLARIES

ARTICLE IV

OF THE PROGNOSIS OF THE DISEASES OF THE HEART

When the diseases of the heart assume an acute or chronic character, the prognostic is always very perplexing Nevertheless there are important modifications to make to the general proposition which I have just announced

In order to establish, with exactness, the prognosis of the diseases of the heart, it is necessary to distinguish these diseases into several kinds they may be divided, (1) into acute, (2) into chronic organic, and (3) into organic properly called

1 The acute diseases of the heart cannot with propriety be inserted among organic diseases, those which come under this denomination, are acute *pericarditis,* and *carditis,* or inflammation of the substance of the heart

In respect to the prognosis, these diseases follow the same order as do all acute inflammations in general, the greater danger that they bring with them, in many cases, is the only point in which the prognosis differs It generally varies as do the same degrees of these inflammations

When acute *pericarditis* is not announced in the beginning by strong symptoms, or severe accidents, and a marked disturbance in the action of the heart does not signify that the organ itself is sensibly affected, and the contiguous viscera, as the lungs, etc seem not to participate of the inflammation, when the subject is besides, sound and well organized, then a pretty favorable prognosis may be formed, hence it is not extraordinary to see *pericarditis,* which is otherwise one of the severest affections, attain, by the combined efforts of nature and art, to a happy termination

But the cases in which the solution of the disease is satisfactory, are not the most common, it seldom happens, therefore, that this inflammation is found distinct from those of the pleurae costales, diaphragmaticae, mediastinae, pulmonales, and from the same affection of a greater or less portion of the substance even of the lungs, and of the surface of the heart itself, which in every instance, is more or less inflamed, then, the disease

either terminates in death, or is transformed into one of those alterations which I have designated under the name of *chronic organic*, according to the purulence of the pericardium, the adhesion of this membrane to the heart, its chronic inflammations, etc , etc

The prognosis of the inflammation of the texture of the heart, or of *carditis* united with the same affection of the other viscera of the thorax, or destitute of complications, is always most perplexing, not to say mortal, in all cases Seldom, therefore, does the inflammation of the parts, whose muscular substance constitutes the basis, obtain without its terminating in suppuration, and the suppuration of the organs contained in the great cavities of the body is generally mortal

It is my belief that acute *carditis* has never been seen to reach a perfect solution, and when cases are quoted, all doubts are not removed, some very justly remain as to the actual seat of the inflammation, which cannot invariably be well ascertained

Hence this inflammation almost always terminates fatally, but the death which it usually occasions may happen instantly or somewhat slowly Thus *carditis* has been known to become fatal in a very few days, while in other instances, when the disease has attained to its highest degree, the most alarming symptoms partially disappear, and a sort of convalescence is established, sometimes even the patient is restored to apparent health, he then flatters himself with a near and perfect cure, but the more intelligent physician perceives only a transformation, or degeneration of the disease into another affection slower, but not less severe, as *a chronic organic* disease is then established, mortal in all cases

Among the acute lesions of the heart, considered relatively to the prognosis, ought to be inserted the partial ruptures mentioned in this work, and the rupture of a fleshy pillar of the heart, and of the valvular tendons

The rupture of the fleshy pillars of the heart (Case XLIV), seems to assume all the most sensible characters of an acute disease, this is at least the inference that must be drawn from the consideration of the assemblage of accidents to which the courier became the victim

The prognosis in the cases where this lesion is manifest, will therefore be desperate, and the physician must announce the fatal event of the disease, which occasions death sooner, as the lesion happens suddenly in a sound organ

The rupture of the valvular tendons appears, according to my observations, to be not so severe and so immediately fatal as that of a portion of the muscular substance Hence in almost all cases in which this rupture has been observed, an organic disease of the whole of the heart has invariably followed, without any acute affection of this organ

The prognosis of the entire rupture or laceration of one of the cavities of the heart is here omitted, such accidents are hardly ever known but from their effect, which is sudden death, or at least exceedingly quick

2 The diseases of the heart to which I give the name of *chronic organic*, are almost all from the effects, consequences or degenerations of acute inflammations whose prognosis has just been mentioned, of this number are the serous or purulent effusions into the pericardium, the adhesions of this membrane to the heart, the ulcerations of the surface of this organ, its chronic inflammation, the softening of its texture, etc

These various affections are almost all, let it be repeated, the results of acute inflammations of the heart Thus, when *pericarditis* has reached so far that the symptoms usually become more moderate, the disease seems sometimes to lose its intensity, but it is evidently protracted farther than there was reason to expect, then the affection acquires different characters which, on account of their being less severe, are not less embarrassing to the eye of the experienced physician The prognosis, at first uncertain, though always dangerous even in the beginning of the disease, less detrimental when the inflammation carried to its highest degree, is suddenly moderated, it becomes more and more unfavorable when the concurrence of particular signs announces that the disease is mistaken, that it degenerates, that a serous or purulent effusion is formed in the pericardium, or the disease assumes some other troublesome termination

If the physician has not been able to decide as to the danger of the affection which has preceded that whose prognosis he wishes to establish, the inquiries which he will be obliged to make for the purpose of gaining a knowledge of the disease, will teach him also what prognosis he ought to form

3 If, in order to treat of the prognosis of the diseases of the heart, I had divided them into curable and incurable, among the first, might have been inserted with the acute inflammations, most of the diseases *properly called beginning organic*, which would undoubtedly yield to care, and medical aid, were the first symptoms of these diseases, to the patients themselves, sufficiently evident and strong to induce them to apply for assistance on the first appearance of the disease, for as it has already been said, there are physical and moral signs, by the benefit of which the experienced and attentive physician may well suspect their formation

But, if these organic lesions are old, if they have made evident progress, if all the functions which are connected with the circulation, suffer already from its alteration, then the prognosis is altogether desperate, the physician has no longer to estimate the danger of the disease, whenever he ascertains its existence, he recognizes a mortal affection, and his experience can enlighten him only in estimating the time that the patient will be able to lead a lingering life, and in the choice of the means capable of rendering it the most supportable

It is from the character, intensity of the organic lesion, the constitution of the individual, his manner of living, etc, etc, that the physician can

pronounce concerning the fatal, near, or more or less remote, period of the subject exposed to his observation

If the attack of the organic disease has been sudden, if, from the beginning, it has assumed some dangerous symptoms, if a very great disturbance of the circulation announces a deep lesion of the principal organ of this function, the prognosis will be far more troublesome than had the disease been more moderate in its attack, and presented different or opposite symptoms

Relatively to the constitution of the individual, if it is vigorous, if the subject is in the flower of his age, if he is free from the various degenerations of the humors, without violent passions, obedient to good advice, etc it will be found that the termination is not so soon fatal as in the contrary conditions Finally, as to the manner of living, if the patient is devoted to vice, debauchery, and every kind of excess, if, from his condition, he is exposed to hard labor, to the inclemency of the atmosphere, to laborious exercise, and to lively moral affections, etc, it will hasten so much the end of his life, while by the means of sobriety, temperance, and care, he will not only prolong his days, but will be able even to prevent, for years, the organic disease, to which sooner or later he will fall a victim

1812

WILLIAM CHARLES WELLS

ONE OF THE EARLIEST CLINICAL REPORTS ON THE CARDIAC PARTICIPATION IN RHEUMATIC FEVER

WILLIAM CHARLES WELLS

(1757-1817)

WILLIAM CHARLES WELLS was born in Charleston, South Carolina, on May 24, 1757 His parents had emigrated from Scotland in 1753 His father was a bookseller who was also skilled in the art of bookbinding As was the custom among Colonial booksellers, he owned a printing press and later undertook to publish a newspaper The Wells family was firm in its loyalty to Great Britain In keeping with the ideals of the Southern gentry, young William was sent across the sea to gain his preliminary education He attended grammar school in Dumfries, Scotland, for two and a half years At the end of that time he spent one year as an undergraduate in the University of Edinburgh

In 1771, Wells returned to his native city and became apprenticed to Dr Alexander Garden Dr Garden was not only Charleston's most prominent physician but also had a deep interest in zoology and botany He was a member of the Royal Societies of Upsala and London, and corresponded in Latin with Linnaeus, who perpetuated Garden's name by the gardenia, which he called "one of the most beautiful flowering shrubs in the world"

With the outbreak of the War of Independence, Wells, whose sympathies were with the King, left for England to be with his father, who had returned to England a few months previously In 1776 young Wells began the study of medicine at the University of Edinburgh In 1778 he passed his preliminary medical examinations He then went to London and there attended the lectures on anatomy and midwifery given under the able direction of Dr William Hunter Later he spent some time at St Bartholomew's Hospital and then studied in Leyden for three months Wells returned to Edinburgh and in June, 1780, received his medical degree

To settle his father's business, which apparently had been mismanaged by an elder brother, Wells embarked on a trip to the Carolinas When the Tories lost control of Charleston, Wells moved the printing press and stock to St Augustine, Florida There he had no time for the practice of medicine, but became printer, bookseller, merchant, and trustee He also spent some time acting in a local theater company On his return to Charleston, Wells was obliged to spend three months in prison because of some debts his brother had accumulated After more difficulties which arose because of his loyalistic tendencies, he sailed for England in May, 1784

On his return to London, Wells became acquainted with Matthew Baillie,[1] with whom he later became intimate He also numbered among his close friends David Hume, Lord Glenlee, Robertson Barclay and William Lister In 1785 he began the practice of medicine in London

In 1788, Wells became a licentiate of the Royal College of Physicians, and in 1789 became physician to the Finsbury Dispensary, an appointment which he held for ten years In 1795 he was elected assistant physician to St Thomas' Hospital, and in 1800 he was promoted to the rank of physician

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[1]See pp 255-256 biographic sketch of Matthew Baillie

In 1792 he published the important work that has endeared him to ophthalmologists, his "Essay on Vision"[2] Therein he explained why objects are seen singly with the two eyes Because of this contribution, presumably, he was elected to the Royal Society in 1793

Another important work of Wells appeared in 1814 This was his "Essay on Dew"[3] Pleadwell suggested that it was the foundation stone of the modern science of ventilation In 1814, also, Wells was elected a fellow of the Royal Society of Edinburgh.

That Wells anticipated Darwin in recognizing the principles of natural selection is suggested in his paper entitled "Account of a Female of the White Race of Mankind part of whose Skin resembles that of a Negro" This was published in 1818 in his collected works

Wells' classic article on rheumatism of the heart, which it is our privilege to reproduce, was written, it must be remembered, before the time of the stethoscope For this reason alone, his contribution to the study of this disease is remarkable

It seems that David Pitcairn, the prominent English physician, who with Matthew Baillie had proposed Wells for fellowship in the Royal College of Physicians in 1797, with an unsuccessful result, was the first physician to express a knowledge of the relationship of rheumatism to heart disease Matthew Baillie referred to Pitcairn's discovery of 1788, in the second edition of his work on "Morbid Anatomy," published in 1797 Wells also referred to Pitcairn in his article Unfortunately, Pitcairn did not publish his observations on rheumatism and heart disease

On July 29, 1789, Edward Jenner is said to have read a paper before the Fleece Medical Society[4] on "Remarks on a Disease of the Heart Following Acute Rheumatism" It is to be regretted that the manuscript of this paper was lost and that Jenner's observations were never published Wells did not know of Jenner's work on this subject Sir David Dundas[5] noted the disease in 1808 and in the same year, according to Keil, J F Davis made note of a colleague's observations on "Acute Rheumatism Attacking the Heart" Wells' contribution, the first thorough treatise on this disease, was read in 1810 and published in 1812

In 1800, at the age of forty-three, Wells began to suffer from apoplexy The disease incapacitated him for several months However, he placed himself on a limited diet and managed to remain in fairly good health until 1813 His health then became poor from general weakness, associated with shortness of breath and swelling of the ankles Wells noticed in 1817 that he frequently made the sudden and deep inspirations which are suggestive of the disease John Cheyne was to describe a year later

In August, 1817, his physicians, Lister and Baillie, who were also personal friends, abandoned hope for his recovery as did Wells, himself He died in London on September 18 of that year

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[2]Wells William Charles *An essay upon single vision with two eyes, together with experiments and observations on several other subjects in optics*, London, T Cadell, 1792, 144 pp

[3]Wells, William Charles *An essay on dew, and several appearances connected with it*, London Taylor and Hessey 1814 146 pp

[4]Keil, Harry A note on Edward Jenner's lost manuscript on 'Rheumatism of the Heart" Bull Hist Med 7 409-411, 1939

[5]Dundas David An account of a peculiar disease of the heart, Med & Chir Soc 1 36-46, 1808

ON RHEUMATISM OF THE HEART*

By

WILLIAM CHARLES WELLS

DR DAVID PITCAIRN, about the year 1788, began to remark, that persons subject to rheumatism were attacked more frequently than others, with symptoms of an organic disease of the heart Subsequent experience having confirmed the truth of this observation, he concluded, that these two diseases often depend upon a common cause, and in such instances, therefore, called the latter disease rheumatism of the heart He communicated what he had observed to several of his friends, and to his pupils at St Bartholomew's Hospital, to which he was then Physician, but no notice, I believe was taken of his remark in any book, before it appeared in the second edition of Dr Baillie's *Morbid Anatomy,* which was published in 1797 No similar observation, as far as I know, is to be found in any book written before that time Morgagni, indeed, and Dr Ferriar of Manchester, had given cases of rheumatism existing with an organic disease of the heart, but it is evident that they considered the concurrence of the two diseases as merely accidental, and it is very probable, that similar cases occur in other authors who wrote before Dr Baillie, though I have not met with them

Since the appearance of Dr Baillie's work, this disease has been treated of by Dr Odier, of Geneva, in his *Manuel de Medicine Practique,* printed in 1803, and by Mr David Dundas, Sergeant Surgeon to the King in a Paper lately published in the *Transactions* of the Medical and Surgical Society of London Dr Odier's work is only a text-book for Lectures given by him on the Practice of Medicine, and as is common in such works, very few references are made in it to other authors As he received, however, his professional education in this country, and has long conducted the medical department of the Bibliotheque Britannique, a literary journal printed in Geneva, he could not be unacquainted with Dr Baillie's *Morbid Anatomy* Mr Dundas takes no notice in his paper of what had been said upon the subject of it, either by Dr Baillie or Dr Odier He could scarcely, indeed, have seen the *Manuel* of Dr Odier, and it is probable, that he had not read the account, which was given of it in the *Edinburgh Medical Journal,* for October, 1806, but there is a greater difficulty in sup-

*Read April 3 1810 Published in Tr Soc Improv Med & Chir Knowledge 3 373-424, 1812

posing, that he was ignorant of what had been mentioned, twelve years before, in Dr Baillie's popular work, respecting Rheumatism of the Heart, on the authority of Dr Pitcairn

As I knew that Dr Pitcairn did not mean to publish anything upon this disease, and as I had good reason to believe, that it was unknown to many practitioners of medicine in this country, I formed the design, about four years ago, of offering a Paper upon it to this Society But very shortly after, I saw the article in the *Edinburgh Medical Journal,* which relates to Dr Odier's book, and, in consequence, determined not to proceed, till I should see the work itself In the meantime, Mr Dundas's Paper appeared, and this I found to contain so much information upon the subject of which it treats, that I necessarily regarded the value of what I had collected myself as much diminished, and therefore abandoned the design of communicating it But considering afterwards, that even a repetition of what had been already said might be useful, in exciting the attention of physicians to a disease hitherto little spoken of, I lately resumed my intention, and now offer, in the form of Cases, what I have acquired from other sources than Mr Dundas's Paper, as our knowledge of the disease is still too imperfect, to admit the formation of a just history of it I once expected that my Paper would be enriched by the contributions of my late most excellent friend Dr Pitcairn, for although I knew that he had preserved no account in writing of what he had seen in this disease, yet I was confident, that the extent of his observation, for he had treated more than a dozen cases of it, and the accuracy of his memory, would enable him to afford me much valuable information But I neglected to obtain this while the opportunity existed, and I now lament my indiscretion

CASE I

Mr T M came from Scotland in April, 1798, to reside in Berkshire, being then in his eighteenth year He was of a fair complexion, short stature, and a habit rather full than muscular From the age of nine years he had been every year attacked with acute rheumatism Four of the attacks had been very severe, each of them confining him to bed for several weeks, the others seldom kept him at home longer than a week, though the redness, swelling, and pain of the joints did not leave him for two or three weeks more While he was labouring under this disease, the pains often shifted in the most sudden manner, and, in the greater fits of it, he was often distressed with a sense of oppression in his chest, frightful dreams, and despondency of mind In November, 1797, he had likewise had a slight spitting of blood

Four weeks after he came to Berkshire, he fell into a small pond of water, while attempting to leap over it, and wetted his lower limbs as

far up as the middle of his thighs He pursued, however, his exercise, and suffered his clothes to dry upon him The following day, while walking in the streets of Oxford, he was suddenly seized with trembling and coldness, principally affecting his lower limbs, with faintness, giddiness, sickness at the stomach, and a sense of oppression in his chest He afterwards became warm, and then began to feel a palpitation of his heart, and a beating in his head In the progress of his illness, he was frequently attacked with breathlessness, a sense of choking, and a feeling as if he were about to expire In the night time he used to be warm, and to sweat After he had been affected in this way about three weeks, he came to London and consulted me At his first visit, I did not become acquainted with all the circumstances which I have mentioned, and as I found his pulse frequent, and tongue white, and was told by him, that he was worse every other day at ten o'clock in the forenoon, I thought it probable, that his disease was a tertian fever, which had not yet fully intermitted As I learned, however, when I saw him next, that the beating in his chest was never absent, though at some times much greater than at others, and that he had been much subject to rheumatism, I began to suspect, that his disease might be rheumatism of the heart, of which I knew nothing, except what I had learned from Dr Baillie's publication I carried him therefore to Dr Pitcairn, who confirmed my conjecture, and was fearful that he would not recover

Mr M went again into the country, but I had frequent letters respecting him and once visited him there I think it, however, unnecessary to say more upon his case, than, that, after he had laboured under the palpitation four months, he was attacked with pains, swellings, and redness of his joints, which continued about six weeks, but were not so severe as to confine him to bed, that during this time the palpitation began to lessen, but that it did not entirely leave him before the end of the second year from its commencement Mr M during his illness was several times seen by Dr Bourne of Oxford He was seen also by Mr James Russell, of Edinburgh, who had often attended him in sickness in Scotland, and having been called by business to Birmingham, had afterwards extended his journey to Berkshire to visit him

Since his recovery I have met with him frequently, and have several times applied my hand to the region of his heart, without feeling there any unusual beating But he says, that exercise is now more apt to excite palpitation than formerly, and that he sometimes experiences it without any apparent cause He thinks too, that it occurs oftener while he is affected with rheumatism of the joints, which continues to attack him every year, than at any other time Before the palpitation comes on, he is seized with a gnawing pain in the region of the heart, and a sense of suffocation In two or three minutes these symptoms either

disappear, or become less, the palpitation then begins, and lasts about the same time Such attacks, however, do not happen oftener than twice or thrice in the year

I may add, that in the course of my correspondence with the relations of Mr M I learned, that one of his uncles, whom he resembles in external appearance, after being severely afflicted with rheumatism, became, when about sixteen or seventeen years old, subject to violent palpitation of the heart, and some time after died suddenly and that, his body being opened, the heart was found enlarged

CASE II

Martha Clifton, aged nearly fifteen years, was admitted into St Thomas's Hospital, on the 18th of February, 1802, after labouring under acute rheumatism about sixteen days Her pulse was small, but the heart struck the ribs with such force, that its beats could be reckoned by applying the hand to the right side of the chest About two or three years before, she had likewise been affected with acute rheumatism, during the presence of which she had been troubled also with a violent beating of her heart In the interval between the two attacks of rheumatism, she had experienced no palpitation in her chest The account of what I did not observe myself I received from the patient, and her mother, but those, who are conversant with the business of an Hospital, know that little dependence is to be placed upon the accuracy of patients or their friends, when they speak of symptoms which have formerly occurred She remained in the Hospital eleven weeks, and was then taken away by her relations, for the purpose of being sent into the country The pains in her limbs were nearly gone, and the palpitation of her heart was much diminished

Many of the tendons of the superficial muscles in this patient were studded with numerous small hard tumours, an appearance I have observed only in one other person, a thin and feeble man forty-one years old, who also laboured under rheumatism

CASE III

Charles Williams, aged twenty years, was received into St Thomas's Hospital the 21st of June, 1804, on account of painful swellings of his joints, under which he had laboured seven months His pulse was quick, and his heart beat forcibly against the ribs He had often, he said, in the course of the last eight years, been affected with a similar disease of the joints, during which he had always been troubled with a palpitation of his heart After remaining a month in the Hospital, without receiving much benefit, he was discharged from it for disorderly conduct

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*Dr Lister has informed me that the superficial tendons of Salmon the subject of the seventh case in this Paper were similarly affected As Salmon did not mention this to me and as I did not discover it myself the same symptom may have existed in several of my patients labouring under rheumatism, besides those of whom I have spoken

CASE IV

Mary Bond came into St Thomas's Hospital on the 9th of January, 1806, labouring under acute rheumatism, which had seized her eight days before She was then in her sixteenth year, had never menstruated, and since her ninth year had been frequently attacked with rheumatism When she had been three months in the Hospital, I discovered, that her heart beat much too strongly, and I was afterwards informed by her mother, that this symptom had always been present, while she was afflicted with rheumatism, but at no other time She staid in the Hospital nearly four months, in the whole, and during that time frequently complained of pains in her chest These, in the month of April, were attended for ten days with cough, difficulty of breathing, and an increase of fever When she left the Hospital, the pains in her limbs were not entirely gone, and her heart was still beating strongly Possibly, however, as the pains of her limbs had lessened considerably in the time between her coming into the Hospital, and the discovery of the palpitation, this symptom had also diminished in the same interval I learned its existence from inquiring, if she was affected with it

CASE V

I visited Miss A L for the first time, on the 17th of September, 1806, at her father's house in Surrey, distant about eight miles from London She was sixteen years old, tall, and thin, and had never menstruated Several of her relations had died of pulmonary consumption, and she herself had laboured under an acute disease of the chest about four years before From that time, however, to the commencement of the train of ailments which I am about to describe, she had enjoyed very good health, and had possessed a much greater degree of bodily strength, than was indicated by her appearance

In the beginning of August, shortly after remaining some time in a cold cellar, she was seized with pains, swelling, and redness of her joints, and fever These symptoms lasted only ten days About a week after they had ceased, she walked about a mile from her father's house, assisted by an attendant, and, while returning, accidentally wetted her feet In the evening of the same day, she was attacked with pains in her feet, which were not accompanied with swelling or redness These pains remained only a day or two, immediately upon their ceasing, her heart began to beat with considerable violence Her right hypochondrium soon after became painful, and about the same time she began to complain of a pain in the tops of her shoulders Various other symptoms had also occurred, but as no regular history of them had been kept, and as the most important existed when I visited her, I shall proceed at once to give an account of the situation in which I found her, at the distance of nearly four weeks from the second attack of external pains

The palpitation of the heart, which had never ceased from its first appearance, was distinctly felt in every part of the thorax, to which my hand was applied. In the arteries, only a shaking was perceivable, which could not be divided into distinct pulsations. The strokes of the heart were one hundred and ninety in a minute; they were equal in force, and the intervals, which were also equal, were so distinct, that I fancied I could have numbered the strokes, if two hundred and fifty, or even three hundred, had been given in the same time. Her breathing was not laborious, and she had no cough; but she frequently complained of a great and indescribable anxiety in her chest. This was always much lessened by her taking a few drops of laudanum and a drachm of vitriolic aether, although no change was ever induced by these medicines on the palpitation. The external jugular veins were swollen, and alternately rose and fell. She had a little headache, was often sick at the stomach, and sometimes puked; what was thrown up was for the most part green, and had a sour smell. Her appetite for food was, notwithstanding, far from being lost. The tongue was somewhat foul, and a small part of its middle was dry, but her thirst was inconsiderable. She had two or three stools daily; their colour had formerly been green, but was now natural. There was now no pain in her shoulders, nor any in her right hypochondrium, except it was pressed. On the day I visited her, the skin and eyes had begun to be a little yellow, and her urine, which was said to be sufficiently copious, now gave a slightly yellow tinge to white linen. On examining her feet, I found them oedematous. Her muscular strength was greater than might have been expected, considering the length and magnitude of her ailments.

I staid all night at her father's, and saw her early on the following morning, when I was astonished at learning, that soon after taking fifteen drops of laudanum, late in the evening, she had become quiet, had remained so the whole night, and had enjoyed much refreshing sleep. The urine, which was passed in the night, had a pink-coloured sediment. In the other symptoms there was no change.

On the evening of the 19th, I visited her again, and was accompanied by Dr John Meyer, of New Broad-street. The day before, she had been thought better; but many things seemed now to indicate her speedy death. The sickness had increased; her face and hands were cold, the skin pale, the motion of the artery at the wrist scarcely perceptible, and the strokes of the heart against the ribs of much less force than formerly. Their number was a hundred and seventy in the minute. She shewed, however, no sign of weakness of mind or of delirium, and her tongue was moist and clean.

My last visit was on the 21st. Shortly after Dr Meyer and I had left her on the night of the 19th, she had vomited a considerable quantity of a thin fluid, mixed with a less quantity of a thick and very black fluid, after which she became better. I could again distinctly perceive motions in the

arteries, though they were not to be reckoned The beats of the heart were one hundred and sixty in the minute, and were felt only in the left side of the chest The skin of the whole body was warm and moist, that of the neck and chest was partially covered with a miliary eruption Her bowels had been several days bound, except when loosened by glysters The abdomen was somewhat swelled, and pressure upon it gave more pain than at my first visit She was drowsy, but this was attributed to some laudanum which she had taken She had lately spitten a little blood, but she was still without a cough Respiration, likewise, was performed with little difficulty, and took place only twenty-five times in a minute

On the following night, as I was afterwards informed, she was restless, which was attributed chiefly to the inflamed state of the skin of the chest, from a blister which had been applied several days before She complained frequently in the night of pains in her legs and feet She took, however, a considerable quantity of food, and her breathings, being reckoned, were found to be only twenty-two in a minute, although there was no diminution in the number of the beats of the heart In the morning, she began to be inattentive to what was passing in her room, and to speak sometimes a little incoherently At two o'clock in the afternoon she died suddenly

I had previously requested a friend of the family to apply, when death should occur, for permission to me to inspect the body Application was accordingly made, and permission obtained But some mistake was committed with respect to informing me of what had happened, and the weather being warm, and the family anxious to have the examination over, this was performed by the apothecary who had attended the deceased, assisted by his partner, and another medical gentleman Had I been present, the younger Mr Cline would have conducted the examination, as he had been kind enough to promise to accompany me for that purpose The following are the principal morbid appearances, which, as I was afterwards informed, were observed

The pleura of the ribs, and that of the lungs, were inflamed, and in many places adhered to each other The lungs felt firm and fleshy, from containing a quantity of coagulated blood The whole of the internal surface of the pericardium was attached to the heart, by means of two distinct layers of solid matter, each having the thickness of a shilling, the outer resembled coagulated blood, while the inner was whitish, and sufficiently tenacious to permit its being torn The surface of the heart was also inflamed, and, from the right auricle to the apex, black, its substance was flaccid, and appeared to be enlarged About a pint of bloody serum was found in each cavity of the chest A considerable quantity of fluid, slightly red, was likewise found in the abdomen The right lobe of the liver was enlarged, and much inflamed, and, on its concave surface, black The stomach, where in contact with the liver, was also black, and many parts of

it had marks of inflammation Many portions of the small intestines were inflamed, and the lower half of the rectum seemed to be gangrenous I must remark, however, that what has been said of the stomach and intestines relates only to their external appearance, for no part of them was opened

CASE VI

John Miller, a sailor aged thirty-six years, pale and thin from bad health, was admitted into St Thomas's Hospital, on the 1st of June, 1809, on account of pains in his limbs, with which he had been afflicted five months His pulse was frequent and rather feeble, and during the two last months he had felt a constant beating in his left side, which upon examination I found to depend upon the action of the heart He remained in the Hospital two months, in which time he became free from pain in his limbs, and in a great measure recovered his flesh, but the pulsation in his side, which had never been very great, continued unchanged He now thought himself sufficiently well to go to sea, and was discharged from the Hospital at his own desire

CASE VII

George Salmon, at present in St Thomas's Hospital, a domestic servant, nineteen years old, of a fair complexion, short stature, and while in health fresh-coloured, and inclined to be fat, but now pale and thin, became the patient of my colleague, Dr Lister, on the 11th of January last From the notes which Dr Lister has been kind enough to communicate to me, and from my own examination of the patient, I have collected the following circumstances of his case

In June, 1808, three days after being heated in a playhouse, and drinking while in this state a considerable quantity of porter, he was attacked with stiffness, pains, and weakness in his limbs, and with pains and swellings in the joints of his fingers During this illness he also laboured, for a fortnight, under a pain of the right side of his chest, and a cough In the course of three months the ailments in his joints became much less, but he did not entirely recover his health for nine months more, during which time he used to feel a beating in his forehead after running, and often had the joints of his fingers swelled for three or four days He remained perfectly well to the middle of December, 1809, when he was seized with stiffness, and slight pain in his lower limbs, attended with a rash, and feverishness The rash occupied various parts of his skin in succession, but was never very extensive, and receded in a week It has since been several times present for a day, but has not appeared for the last six weeks Shortly after the beginning of his illness, the joints of the fingers began to swell again, and to be painful when pressed, in which state they still continue Sometimes they are slightly red, and the back of his right hand was lately swelled, and a little red, for a few

days The stiffness and pain of his lower limbs began to decrease soon after he came into the Hospital, the former is now nearly gone, and the latter has not been felt by him for a fortnight In the first part of his stay in the Hospital, he was attacked with a pain in his left side, which was increased when he drew his breath, this remained about fourteen days, and he has lately been frequently troubled with a slight cough Since his admission into the Hospital, it has been discovered, that his heart palpitates He does not know that he laboured before under this symptom, but this seems no proof of its not having existed, for he is still scarcely ever conscious of it, from any internal feeling While he is sitting or lying, the palpitation is often not to be perceived from applying the hand to his left side, but as soon as he rises it becomes very evident The pulse varies in point of number very considerably, but is generally between ninety and a hundred The strokes are full, but are easily made to vanish by pressure Each stroke is given rapidly, as if with a jerk, forming, I think, what Morgagni calls the vibrating pulse, and which so frequently occurs in diseases of the heart, particularly at their commencement, and in acute rheumatism His skin is cool, but at night, he says, his feet often burn His tongue is a little white, his appetite much diminished, his bowels open, his urine is of a deep straw colour, but does not become turbid by cooling He has no difficulty of breathing, except what he attributes to weakness, and his head is free from pain and uneasy feelings He sleeps little, but is unable to assign any reason for it*

Having finished the description of the cases seen by myself, which I think may be properly arranged under the title of rheumatism of the heart, I shall next relate several more, the knowledge of which I have derived from other sources

CASE VIII

This has been furnished by Dr Baillie, and will be given in his own words

"March 25, 1807

"A boy about ten years old, of a fair complexion, and irritable constitution, who had a scrophulous scar under the left side of the lower jaw, after labouring many months under rheumatism, was attacked with palpitation of the heart, and some time after died He was attended by Dr Vaughan, Dr Reynolds, and myself His body was examined, but I was not present The heart, as I have been informed, was somewhat enlarged, and there was a strong adhesion of the pericardium to it He had a few tubercles of the lungs, and I believe some of the glands of the mesentery were enlarged The liver was also of a greater size than usual "

*This patient left the Hospital a day or two after his case was taken by me

In another communication, dated in April, 1809, Dr Baillie says—"I have known a good many instances of palpitation of the heart in children, and young people of both sexes I cannot, however, now remember distinctly more than three or four, where this affection was preceded by rheumatism But I can hardly doubt that several cases of this kind have been forgotten by me"

CASE IX

I received this from my colleague, Dr Lister

"Miss P--, thirteen years of age, became my patient on the 17th of May, 1807, at which time she laboured under a very considerable difficulty of breathing, and a palpitation of the heart so violent, that not only the motion it gave to her clothes might be seen at a distance, but her body itself was shaken by it Both these symptoms were increased by the least exercise She had a slight cough, her countenance discovered great uneasiness, her pulse beat a hundred and thirty-six times in a minute, the tongue was white, the appetite was less than natural, the bowels were confined, the urine was in the usual quantity She was emaciated, and her emaciation was said to have taken place during her present illness I was informed, that she had been attacked in the beginning of the preceding February with acute rheumatism, which lasted about a fortnight, and that, when this left her, the shortness of breath, palpitation and cough came on At first the cough was very considerable Her feelings became less uneasy, and her pulse slower, under the use of a spare diet, and of a blister applied to the region of the heart The alleviation of symptoms occurred too quickly to have been the effect of digitalis, which she was taking at the same time On the 25th, the feet began to be oedematous, on the 29th the abdomen was swelled, and a fluctuation was to be felt in it On the 5th of June, the anasarca was general, the swelling of the abdomen was increased, the pulse intermitted, and was slower On the 6th, in the morning, the swelling of the abdomen was lessened, and the patient thought herself much better In the evening, she was suddenly seized with extreme difficulty of breathing, and an occasional suspension of breathing, while the breathing was suspended, the pulse either did not beat at all, or beat very slowly On the 7th, at two o'clock in the morning, she died

"Leave having been obtained for examining the body, the examination was made by my friend Mr Smith, of Southampton-street, on the 8th of June, and the following is the account he was so good as to give me of the appearances he observed

"The lungs adhered to the pleura costalis almost at every part The left side of the chest contained about five ounces of water, in the right side there was about one ounce The pericardium adhered to the whole surface of the heart, the adhesion was easily separable by means of the fingers The heart was twice as large as natural, its muscular structure was increased in

thickness, and all its cavities were very much loaded with blood The cellular membrane of the lungs contained some water In the cavity of the abdomen there was about a pint of water, in which were floating several portions of coagulable lymph The viscera of the abdomen were free from disease "

CASES X and XI

Both of these have been communicated to me by Mr Benjamin Brodie, Assistant-Surgeon to St George's Hospital

"A girl, fourteen years of age, was admitted into St George's Hospital, in the middle of April, 1807, with symptoms of acute rheumatism affecting the extremities These symptoms in a short time subsided, but were immediately succeeded by pain in the chest, attended with a sense of oppression in breathing, palpitation of the heart, a quick feeble pulse, and general debility On the 23d of May she died

"On inspecting the body, the lung on each side was found adhering to the pleura lining the ribs, but the adhesions were not of a recent date

"About twelve ounces of serous fluid were effused into the cavities of the chest

"The pericardium was much inflamed, and the two folds of that membrane were united by a layer of coagulable lymph "

"A woman, twenty-five years of age, was admitted into St George's Hospital, under the care of Dr Nevison, in July, 1807, labouring under dropsy of the abdomen, anasarca of the lower extremities, and a constant palpitation of the heart She said, that some months previous to her admission she had been attacked with a rheumatic fever, that, on the fever subsiding, she was seized with the palpitation of the heart, which had continued ever since, and that the dropsical symptoms had appeared more lately She died a few weeks after her admission

"On inspecting the body, the lungs were found partially adhering to the mediastinum There were adhesions every where between the two folds of the pericardium On the internal surface of the left auricle of the heart, there was a space, of about an inch square, studded with very minute excrescences resembling small warts Three excrescences of a larger size were found on the internal surface of the left ventricle, about an inch below the semilunar valves One of these was so large, as to project about half an inch into the cavity of the ventricle Two or three similar excrescences were attached to the mitral valve, and semilunar valves of the aorta "

CASE XII

This case was originally published in the *London Medical Journal,* for April, 1803, by Mr Wagstaffe of Southwark, in whose practice it had occurred As it appears to me valuable, for this, among other reasons, that

the body was examined after death by a teacher of anatomy, Mr John Taunton, I shall here give an abridgment of the original account of it

Miss M aged about fourteen years, of a spare habit, sallow complexion, and active disposition, became the patient of Mr Wagstaffe, in the middle of January, 1802, on account of her labouring under acute rheumatism After continuing ill in town for nearly two months, she went into the country, whence she returned in a short time, apparently in good health She remained well till the beginning of October, when she was again attacked with acute rheumatism This disease disappeared in about a week, leaving a most distressing cough, an excruciating pain in the left side, palpitation of the heart, and difficult respiration, attended with great dread of suffocation The pulse varied from a hundred to a hundred and forty in the minute, it was sometimes throbbing, at other times weak Blood taken from the arm, at this period of the disease, exhibited but slight marks of inflammation In a few days the respiration grew more difficult, and the patient now began to be sometimes affected with vomiting Her situation afterwards became much less distressing, apparently in consequence of medical treatment, but the amendment did not continue long, and she died in great agony on the 23d of November

On opening the body, strong and extensive adhesions were found between the lungs, and the adjoining parts, but the lungs themselves were sound The pericardium was attached so closely to the heart, that it was very difficult in most places, and in some quite impossible, to separate them The heart was enlarged, but its structure was natural The abdominal viscera were in a healthy state

The preceding cases appear to me just instances of rheumatism of the heart The two which follow are less so, but seem, notwithstanding, sufficiently connected with my subject, to excuse my relating them

Philip Smith, aged fifteen years, feeble from his birth, became a patient in St Thomas's Hospital, on the 9th of July, 1807 In 1804, he had laboured under acute rheumatism four months Two years after this, he was seized with a fever, which lasted also four months During the fever, his heart began to beat more strongly than formerly, and it continued to do so ever after From the time of his recovery from the fever, he had often felt pains, chiefly at night, in his lower limbs The front of his head almost constantly ached, and blood had twice lately flowed from his nostrils His face was frequently flushed, and sometimes appeared to him a little swollen After he had been a fortnight in the Hospital, he was attacked with a pain in his right side, and a cough He was an only child, and his father was so unhappy while they were separated, that he took him home on the 30th of July

Mr E of Canterbury, about twenty-four years of age, of a fair complexion, and stout make, after being long constantly afflicted with rheu-

matism, either in its acute or chronic form, became subject to attacks of extreme difficulty of breathing, and a sense of tightness across his chest, attended with a pulse always frequent and small, and sometimes irregular. These attacks had of late occurred about once a month. They were frequently preceded by a slight inflammation of the fauces, and were sometimes accompanied with inflammatory swellings of the joints, but never with cough, or pain in the chest. When the difficulty of breathing was most urgent, no motion was perceptible in the abdomen from respiration. Such was the account of Mr. E.'s disease, which I received in September, 1806, from his medical friends in Kent. By my own examination of him I learned, that in his best state he had a constant uneasiness under his sternum, which was increased by a deep inspiration, that he had often a beating of his heart while he was at rest, and always after he had walked a little quickly, when it was attended with breathlessness, that his pulse was frequent, and that his urine was highly coloured when first made, and became turbid on cooling. A month after he consulted me, I was informed by one of his medical friends that he was better, and the following year I was told, by a person whom he sent to me, that he was altogether well.

I think it proper also to mention here, that I have seen four persons die of peripneumony, which had supervened to acute rheumatism. The heart of one of them, a sailor boy, fourteen years old, beat with more than ordinary force, while he laboured under the disease of his chest. In another, a female servant, nineteen years of age, a patient of Dr. Lister's in St. Thomas's Hospital, the heart palpitated strongly in the beginning of the peripneumony. Her body being opened, along with other marks of disease in the contents of the chest, the pericardium was found to adhere in various places to the heart. The heart was not enlarged, but its muscular substance was in several places inflamed. Permission could not be obtained to examine the bodies of any of the other three patients. In all the four, the disease of the limbs either had become less, shortly before the accession of peripneumony, or was considerably diminished very soon after. Several other examples have been seen by me of an attack of peripneumony, in persons affected with acute rheumatism, but in these the patients recovered.

To render the historical part of my subject more complete, I shall add, that, in Mr. Burns' *Observations on the Diseases of the Heart,* there is an account of a girl affected with palpitation, who, among other symptoms, had frequently shifting pains in several of the large joints, and had formerly laboured under rheumatism, that, in the Nineteenth Number of the *Edinburgh Medical Journal,* an instance is related by Mr. Crowfoot of acute rheumatism, in a tall feeble man, about twenty-two years of age, being attended with symptoms of a diseased heart, and that of the three cases of carditis, which have been published by Dr. Davis, in his treatise on that disease, the first occurred in a girl twelve years old, who had laboured eight

days under pains in her left shoulder, and insteps, before any symptom of a diseased heart appeared, and became free from those pains the second day after the accession of such symptoms, the second, in a boy seven years old, who suffered acute pains in the lower extremities, during different parts of his illness, and the third in a girl sixteen years of age, the disease of whose heart succeeded the sudden disappearance of inflammation in her feet and ancles

In regard to the treatment of rheumatism of the heart, the propriety of enjoining rest and low diet in the beginning of it, and, in every stage, of producing a discharge of serous or purulent matter from the integuments of the thorax in the neighbourhood of the heart, by the use of cantharides or other means, will, I believe, be admitted by every person But, when the tender age of those most liable to it, and their frequent weakness, whether original, or consequential to the disease of the joints which had previously existed, are considered, it may often appear improper to bleed, even at its commencement Besides, as it has been found, that in London bleeding is never necessary for the cure of acute rheumatism of the external parts, and sometimes proves highly injurious, and, as the translation of the disease to the heart seems analagous to the recession of gout from the extremities, additional arguments may hence be derived against the general practice of bleeding, even in the very onset of the disorder My own opinion, however, is in favour of copious bleeding, in the beginning of the disease, notwithstanding the force of the arguments which I have related When the disease of the heart has quickly followed the entire disappearance, or considerable diminution, of that in the joints, we may attempt to bring back the latter, or to imitate it, by inducing inflammation in their integuments I followed this practice in the case of Miss A L , but she suffered so much distress from the stimulating substances, which were applied to her joints, that her mother soon removed them, and, as her situation had from the first appeared to me hopeless, I thought it cruel to urge their renewal If the disease assumes a chronic form, and there be evident signs of an enlarged heart, it should, in my opinion, be treated, as if the enlargement had never been connected with external rheumatism, in which case, Dr Ferriar of Manchester has experienced beneficial effects from the use of tonic remedies

What has hitherto been said of the method of cure relates, chiefly, to the most considerable cases of the disease In others of less magnitude, a different mode of treatment may sometimes, perhaps, be with propriety adopted, especially if they have been of long standing In the second case, for instance, related by me, that of Martha Clifton, as I was informed that she had laboured under palpitation of the heart, in a former fit of rheumatism of the limbs, and that both diseases had left her at the same time, I applied myself solely to the removal of that in the limbs, expecting that the

other would recede with it, and the event partly justified my practice, for they diminished together, though they both existed in some degree when she left the Hospital I received similar information regarding the cessation of the internal and external disease in the former attacks, which had been suffered by the subjects of the third and fourth cases, and followed therefore a similar mode of practice On the same grounds, I employed mercury in the treatment of John Miller, the subject of the sixth case, but unsuccessfully with respect to the removal of the palpitation

To conclude, I take the liberty of calling to the recollection of the Society, that the bodies of six of the persons, whose cases I have related, were examined after death, and that in two of them the liver was found diseased I had supposed this to arise from the impeded passage of the blood from the vena cava through the heart, and its consequent congestion in the vessels of the liver, but I have lately learned, that Dr Odier has seen rheumatism translated from the joints to that viscus The two instances, therefore, of disease in the liver, to which I have referred, may possibly have arisen from a common cause with the disease of the heart and not have been the effect of it *

POSTSCRIPT

Read November 5, 1811

After the preceding Paper had been read to the Society, two further cases of rheumatism of the heart occurred to me, which seem worthy of being made known to it

CASE XIII

Charles Mills, aged sixteen years, was admitted into St Thomas's Hospital, on the 17th of August, 1810, after labouring three days under pains of his limbs He was of a feeble appearance, and, four years before, his right leg had been amputated, in the same Hospital, on account of a long disease of the ancle I saw him first at one o'clock in the afternoon of the 18th His ancle was swelled, painful, and a little red His pulse was frequent, and his face flushed, and both the latter symptoms were in a greater degree, as I then thought, than could be occasioned by the disease of the ancle, though he complained of nothing else Three hours

*The following case which I met with after the preceding Paper had been read to the Society, strengthens my former opinion

A sailor boy, seventeen years old, was received into St Thomas's Hospital, June 6, 1811, *after he had laboured four months under a pain in the region of his heart* difficulty of breathing and a slight cough His pulse was frequent his heart beat somewhat too strongly especially when he stood or walked and his lower limbs were a little dropsical He remained nearly in the same state till about the *middle of August All the symptoms* of the disease in his chest then increased and he died on the 2d of September I had never observed the beats of his heart and arteries to have, during any short space of time unequal force, or to follow one another at unequal intervals No disease of the limbs had preceded or accompanied that in his chest His body was examined the day after his death The pleura of the lungs adhered in many places to the pleura of the ribs, and between other parts of those membranes were found about twelve ounces of a watery fluid The heart was enlarged, but not considerably The pericardium and the *covering of the heart adhered every* where so closely together that they could not be separated and scarcely any line of distinction could be perceived between them The aortic valves [illegible] other respects the heart was sound Both the liver and spleen [illegible] substance appeared to be without disease The stomach and [illegible] large A few ounces of a watery fluid were found also in the abdomen

afterwards, he was attacked with a pain in his left side, difficulty of breathing, and a slight cough. In two hours more, the pain in his side was increased, but that of the ancle was nearly gone. At nine o'clock in the evening, the pain of the side, and the difficulty of breathing, having become greater, and his heart having begun to palpitate strongly, six ounces of blood were taken from his arm. After the bleeding, the symptoms were less for half an hour, at the end of which time they became as considerable, as they had been before. At four o'clock the next morning, the pain in the side was very great, and the palpitation violent. The patient frequently nearly fainted, and his pulse was one hundred and thirty in a minute. Eight ounces of blood being now taken away, the palpitation and pain became less. A blister was soon after applied to his left side. The occurrences after one o'clock on the 18th took place, while I was absent from the Hospital. I saw him again at one o'clock in the afternoon of the 19th. His breathing was then a little difficult, and his pulse one hundred and twenty in the minute, small, and hard, but the palpitation had ceased entirely, and the pain of his side had nearly ceased. On the 20th, his pulse was one hundred and twelve, and was softer and fuller than on the preceding day, he had no pain either in his side or ancle, and no cough. On the following day the palpitation was present several hours, and he complained of a feeling of tightness across his chest. Eight ounces of blood were in consequence taken from him, and it was directed, that the blistered part of his side should be dressed with the ointment of cantharides. From this time I scarcely ever observed the palpitation to be entirely absent. On the 28th his pulse was ninety-two, to which it had gradually fallen. He complained this day of a pain in the right side of his chest, and on the 4th of September of pain in his ancle and left shoulder, but, in both cases, the pain lasted only a day or two. The palpitation having increased considerably, and the pulse having become more frequent, eight ounces of blood were taken away on the 11th of September, though he had no pain in his chest. During the whole of his stay in the Hospital, he had been restricted to a low diet, and had taken as much tartarised antimony every six hours, as his stomach could bear without sickness being produced by it. When his bowels were costive, a little Epsom salts had been given to him. A discharge of serum or pus, from the skin of the left side of the thorax, had always been preserved. On the 15th of September, the antimonial medicine was omitted, and he was ordered to take ten drops of the tincture of digitalis three times a day. He used the digitalis for more than three weeks, and during this time the pulse was mostly as slow as it is in a healthy person, and sometimes intermitted, but the palpitation of the heart was scarcely lessened in force. On the 11th of October he left the Hospital. I saw him three months afterwards, at which time the strokes of his heart against the ribs were more frequent, and much

stronger, than they ought to have been, if he had been entirely well His external appearance, however, was nearly that of a healthy person He said, that he had now no ailment, and that, for the most part, he did not feel any beating at his heart, but that shortly after he went from the hospital he had been seized with a pain in his chest, which, however, left him soon, without his using any medicine I saw him again a few days ago, about a twelvemonth after he went from the Hospital He has become taller, and more robust, and has the look of being in perfect health, and this he said he enjoys But I found his pulse to be one hundred and ten in a minute, and his heart to beat strongly against the ribs The beating he imputed to his surprise at seeing me, and he assured me, that now he almost never experiences the slightest degree of it As I staid with him, however, half an hour, and during the whole of that time perceived alteration in the action of the heart, I must conclude, at least, that a small degree of surprise produces a greater effect upon it, than would happen, if there did not exist in it some remnant of an organic disease

CASE XIV

Anne Warwick, a nursery maid, in the twenty-first year of her age, was received into St Thomas's Hospital, on the 14th of March, 1811, being then affected with acute rheumatism, which had attacked her a month before She had also pains in her chest, which I supposed to be seated in the external muscles, and a headach In the course of the two preceding years, she had twice laboured under acute rheumatism, in a more considerable degree than at present, but at both those times she was free from ailment in her chest The day after she came into the Hospital, she was seized with a pain in the region of the heart, on account of which a blister was applied to her left side The following day I found her heart to beat strongly, which she said it had done nearly two days Her breathing was also difficult, but she had no cough Ten ounces of blood being taken from her arm, the symptoms of the disease in her chest were lessened, they encreased, however, on the morrow, and again became less, after a blister had been applied over the sternum Her pulse was one hundred and forty in a minute, and she had now no pain in any limb except the right leg On the evening of the 18th, she was breathless, and had a great tendency to faint, but she did not complain of pain in her side or palpitation The disposition to faint was, indeed, always present, when the disease of the chest was urgent, and at such times she frequently did faint Twelve ounces of blood were drawn from her arm, and she soon became better On the 21st, the difficulty of breathing and palpitation returned, but ceased almost immediately after she lost ten ounces of blood I here mean by palpitation such a degree of beating of the heart against the ribs, as excited the patient's attention, and was uneasy to her For I believe, that the heart, from the time of the first attack of the pain

in her side, had always beat much more strongly in her, than it ordinarily does in a person in health She remained free from uneasy feelings in her chest till the 28th, when she was attacked again with pain in the left side, and palpitation of the heart, both of which ceased on the following day, within two hours after twelve ounces of blood were taken from her The pain of her side never returned, but she was often afterwards breathless and disposed to faint, particularly when in an erect posture, but, as her strength had been much reduced by bleeding and other circumstances, these symptoms were probably occasioned only in part by the disease of her heart On the 31st, about an ounce of blood flowed from her nose, soon after which her headach ceased, which had been almost constantly present from the day of her admission, into the Hospital She continued long feeble, and suffered much from various ailments, during the rest of her stay in the Hospital, which lasted till the 1st of the following June When she went away, she complained of nothing, but she was still weak, and her heart still beat strongly One of the nurses of the Hospital saw her about a month afterwards, at which time she appeared to be altogether well I have no doubt, however, but that the action of her heart was then too great

I shall now mention several things respecting this patient, which I have hitherto omitted speaking of, in order that the narration of the chief circumstances of her case might not be interrupted

1 For the first four weeks, the only medicines she took internally were lemon juice, neutralised by salt of tartar, and infusion of senna with Epsom salts During the same time, a constant discharge, either of serous or of purulent matter, from the skin of the left side of her chest, was procured by means of cantharides, but when she became feeble, the discharge was allowed to cease, as the cantharides irritated her then considerably At the commencement of the fifth week, she began to take the tincture of digitalis thrice a day, in doses of twenty drops, and she continued its use eight days It seemed to produce no effect, either upon her pulse or her stomach

2 Her pulse, during the whole of her stay in the Hospital, except upon one day, was very frequent, once, while she was in bed, it was a hundred and forty-four in the minute, but its strokes and intervals were always equal, except on the day to which I have just alluded It was then only seventy-six in the minute, and both the strokes and intervals were very unequal It was felt while she was in a sitting posture, and she had not taken digitalis for five weeks

3 I have said that, in the first attack of the disease of her heart, she had no cough, no cough ever occurred in the progress of that disorder, from which it seems probable, that the inflammation did not affect any portion of the lungs

4 After she had been a month in the Hospital, I discovered an eruption on the skin of her chest, arms, and hands, which I pronounced positively to be the itch It went away, however, in three weeks, without any means being employed to remove it

5 Five weeks after she came into the Hospital, she complained of her throat being painful A few days afterwards, I perceived her breath to smell, as if she were in a salivation from the use of mercury, her tongue at the same time felt sore, and had white spots upon it Saliva also ran from her mouth, though in no great quantity She had used no mercury, in any form, while in the Hospital This state of her mouth lasted nearly three weeks, and caused her to become feeble, chiefly by preventing her from taking food Small doses of Peruvian bark were now prescribed for her, and she was allowed a little porter Hitherto she had been kept on a low diet, and had been debarred the use of all fermented liquors

6 Immediately after her mouth had become well, and as her strength was returning, her feet and legs began to be dropsical, but her progress towards recovery did not seem to be retarded by this event, and the swellings disappeared in the space of ten days While they were present, she took Griffith's mixture of iron, myrrh, and salt of tartar

7 The pains in her limbs, which were said to have nearly ceased at the time she was first seized with the pain in her side, never returned with any violence Indeed, while the disease of her chest was most consider-able, they were sometimes entirely absent They increased a little, when the disease of the chest became less, but left her altogether a few days before she went out of the Hospital

In both of the preceding cases, the general health of the patients seems to have suffered from the means which were employed to overcome the internal inflammation, but, I shall not hence be deterred, from using the same means in an equal degree, in any similar case that may hereafter occur to me The palpitation, which remained in both patients so long after the violence of the disease had been subdued, probably depended, in part, upon some relic of inflammation in the immediate covering of the heart, and, in part also, upon the heart being irritated by the adhesion of the pericardium to it As the palpitation, however, entirely ceased, in the course of time, in Mr T M the subject of the first Case in the foregoing Paper, it is to be hoped, and perhaps expected, that time will produce a similar effect in the subjects of the two last cases

1818

JOHN CHEYNE

DESCRIPTION OF THAT PERIODIC TYPE OF RESPIRATION LATER TO BECOME KNOWN AS THE CHEYNE-STOKES TYPE

JOHN CHEYNE

(Courtesy Medical Classics)

JOHN CHEYNE

(1777-1836)

JOHN CHEYNE was born on February 3, 1777, at Leith, the seaport of Edinburgh. His father was a physician and, according to Pettigrew, was a man of great cheerfulness, benevolence, good sense, and singleness of mind. Cheyne's mother was the daughter of William Edmonston, a fellow of the Royal College of Surgeons (Edinburgh).

Young Cheyne's education began with four years at the grammar school at Leith. When he was ten years of age he was sent to the high school at Edinburgh. There he was placed under the care of Dr. Adam, rector and headmaster. This seems to have been an ill-advised step, for Cheyne was not yet ready for high school and was consequently unable to keep up with the required pace. In general, he was very unhappy while he was at school.

Soon he left high school and was tutored by a clergyman of the Episcopal Church of Scotland. Under his new instructor he studied Greek and Latin for a period of two years, but again, as at high school, he apparently profited little from this contact.

When he was twelve years old he made his acquaintance with the practice of medicine by assisting his father in caring for the elder Cheyne's charity patients. It was his duty to supply these patients with medicine, to bleed them when necessary, to dress their wounds, and to report their conditions to his father.

In 1792, Cheyne began to attend the medical lectures at the University of Edinburgh. His contacts with his father's patients, effects of medical lectures he had heard, the boarding-house jargon of medical students, his frequenting a club of students who alternately examined each other in the required medical subjects, and the assistance of the celebrated ''grinder,'' Mr. Caudlish, all combined to aid Cheyne when he took his examination in 1795. This he passed without difficulty, and obtained his medical degree.

Following graduation, Cheyne left Edinburgh for Woolrich, where the Royal Regiment of Artillery was quartered. There he was appointed to the medical corps as assistant surgeon. He served with the army in various parts of England until 1797, in which year he was elevated to the rank of surgeon. He accompanied a brigade of home artillery to Ireland, and was in action in the campaign against the rebels at Ross and Vinegar Hill in 1798.

Cheyne left the army in 1799 and returned to Scotland. On his return he was placed in charge of the Ordnance Hospital at Leith Fort. He also assisted his father in medical practice. At this time he was fortunate in making the acquaintance of Charles Bell (1774-1842), the leading British physiologist of the day, who later was knighted for his researches in the field of neurology. Bell assisted Cheyne in making dissections and taught him the rudiments of pathologic anatomy.

Cheyne had in 1795 published his first work, ''De rachitide,'' which no doubt was his doctorate thesis. He continued his studies of pediatrics and between 1801 and 1819 published several essays on the diseases of children.

In 1809 Cheyne left Scotland and after visiting Dublin, decided to practice medicine there. His first two years in Dublin were rather unproductive so far as patients

were concerned But in 1811 he was appointed physician to the Meath Hospital, and the appointment seemed to be the turning point in his career He soon was called on to lecture on military medicine at the Irish College of Surgeons, and his private practice began to increase In 1815 he was appointed one of the physicians to the House of Industry

The Dublin hospital reports for 1818 contain Cheyne's classic paper, "A Case of Apoplexy, in which the fleshy part of the Heart was converted into fat" In his historic account, which we are reprinting, Cheyne mentioned the unusual type of breathing now known as the "Cheyne-Stokes respiration" Stokes, as we shall show, put more emphasis on the diagnostic value of this symptom, but Cheyne was the first accurately to describe it

In 1820 Cheyne was appointed physician-general to the Army in Ireland This was the highest medical rank in Ireland With this rank and with a highly successful medical practice, he felt he had fully attained the object of his ambition

Cheyne's health had never been robust and his medical practice fatigued him to such an extent that he soon found it necessary to limit its scope He gradually declined the responsibilities of private practice and limited his work to that of a consultant In 1825, at the age of forty-nine, he was afflicted with a type of "nervous fever" This deprived him of much of his remaining strength and he sought relief by spending a few months in England He later returned to Dublin, where he continued some professional activity until 1831 Then he decided to retire He moved to a country town, Sherrington, in England He did not give up his interest in medicine, but gladly undertook to contribute articles for the "Cyclopaedia of Practical Medicine"[1]

In 1833 a cataract formed in his right eye, depriving him of the sight of that organ, and in 1835 gangrene developed in one of his limbs He died on January 31, 1836

[1]*The Cyclopaedia of Practical Medicine, comprising a treatise on the nature and treatment of diseases, materia medica and therapeutics, medical jurisprudence, et cetera,* edited by John Forbes Alexander Tweedie, and John Conolly, London Sherwood Gilbert and Piper, 1833, 4th vol

A CASE OF APOPLEXY, IN WHICH THE FLESHY PART OF THE HEART WAS CONVERTED INTO FAT*

By

J. CHEYNE

DOUBTS having been entertained of the conversion of the fleshy part of the heart into fat, and only one dissection,† in so far as I know, having been published illustrative of that very curious morbid alteration, the following case and dissection have been thought of sufficient importance to meet the public eye

In this dissection, although no chemical experiment was made in proof of the matter into which the heart was converted being fatty, I have no doubt that it was so Placed along side of the fat which lay over the ribs, I could perceive no difference, save that it was softer and more easily torn, and rather of a deeper yellow, the substance in question communicated a greasy stain to paper, and the animal oil in viscous drops adhered to the knife used in dissecting the heart I was not, at the time of dissection, aware that the morbid change was so uncommon, or that the specimen which lay before me was perhaps the most complete exemplification ever witnessed of the conversion of the flesh of the heart into fat

The patient certainly died of apoplexy, and apoplexy in this case must have depended upon increased action of the vessels of the head The heart itself was apparently incapable of communicating much impetus to the circulating mass

Certainly the dissection would have been more complete had the liver been examined at the same time I may observe, that although the function of the liver had frequently been disordered during the last ten years of the patient's life, I should not have been surprised had that viscus been found apparently sound I am persuaded that diseases of the liver, which do not end in structural changes, often produce the greatest disturbance of the constitution, laying the foundation of fatal diseases of distant organs

A B, sixty years of age, of a sanguine temperament, circular chest, and full habit of body, for years had lived a very sedentary life, while he indulged habitually in the luxuries of the table

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*Published in Dublin Hospital Reports 2 216-223, 1818 We reprint from Medical Classics 3 705-709 1939—F A W 1940

†See a dissection illustrative of this morbid change in an elaborate paper on inflammation of the heart by Dr Duncan Jun See Edin Med and Surgical Journal Jan 1816

This gentleman having had several attacks of the gout in his feet, began a course of magnesia in the year 1813, after which he had only one regular attack of the gout For many years he had been subject to severe attacks of catarrh, which ended without much expectoration He had long been subject to oedema of the ankles in the evening, for two or three years before his death (the time could not be ascertained) he had remarked an occasional intermission in the pulse of his heart

In the latter end of January 1816, he consulted me for a pain in his right side under the false ribs, for which he took calomel at bedtime, and salts in the morning, repeating these once or twice, but he neglected my directions with regard to diet, nay, his appetite being remarkably keen, he ate more than usual, and took at least a pint of port wine or Madeira daily, as was his habit, and this notwithstanding a hard frequent cough, which came on after I was consulted by him

On the third of February he had walked a good many miles and came home exhausted, with a fluttering or palpitation of his heart, for he could not well say which, in a degree he had not felt before He ate as usual, and drank six or seven glasses of wine, which he thought relieved the fluttering He was sitting at tea about nine o'clock when he was attacked with a severe fit of coughing, during which he fell from his chair insensible I saw him in three or four minutes after his fall, and found him with a contusion on the upper and left side of the frontal bone, he was confused, and unable to recollect himself, he was conscious that some accident had befallen him, the exact nature of which he declared himself incapable of understanding His pulse was extremely irregular and unequal It bounded quickly for several pulsations, then it paused and went on more quickly, but with less force He was pale, but none of the muscles were affected with palsy I lost no time in having blood drawn from his arm to the amount of nearly a pound He gradually became more collected, but his pulse continued irregular and unequal, his countenance became flushed, the cough occurred in suffocative fits, and he complained of pain on either side of the tuberosity of the occipital bone Twelve ounces more of blood were drawn about an hour after the first blood-letting, after which the pulse, though it continued equally irregular, was much softer He complained of the contusion, and of considerable pain behind his ears He was removed to bed, the heat of the extremities was restored, and fifteen leeches were applied over the contusion, and he took two pills consisting of two grains of James's powder, three of calomel and four of compound extract of colocynth

On the 4th of February he had several large bilious stools, his understanding was unimpaired, his recollection restored, and he seemed to comprehend the nature of his illness, and he had a sense of fulness in his head, which led me to order him to lose a few more ounces of blood It

would be tedious and unprofitable to particularize the medicines which were ordered from day to day for this patient, they consisted of a mild mercurial every second or third day, and squills with ammoniacum, etc. These were indicated by the loaded tongue, scanty high coloured urine and dry cough. The expectoration being restored, the squills were laid aside on the 15th of February, as they produced nausea and extreme depression of spirits, and bitter infusion with tincture of cardamoms and soda was prescribed. On the 19th a horse-radish bath was ordered, in consequence of some slight demonstration of gout. On the 21st he had some smart pain, with slight inflammation in the ball of the left great toe. About this period he submitted with so much dissatisfaction to a reduced diet, and declared himself so much better after food, that we were induced to allow him a couple of glasses of wine, and to encourage him to take carriage exercise. The irregularity in his pulse never ceased. On the 1st of March he had a return of the suffocative cough and flushing, with some wheezing, which again seemed to demand blood-letting, which was practised with immediate relief. At this period a blister was applied over the region of the heart, which had become the seat of considerable increase of pain, and a discharge was maintained from the blistered surface, by means of ointment of savine and cantharides, about the 4th of March, the sputa became free and concocted. His tongue at this period was for many days furred and of a dark brown colour, as if it had been sprinkled with ground coffee, it was expanded, and its edge was moist. On the 25th of March he began to complain of wheezing, more particularly after exertion, but it sometimes attacked him when he was at perfect rest, his legs and ankles became oedematous, the urine very scanty, much loaded, but without being coagulable by heat. At no period of his illness did his pulse beat more than twelve, or fifteen strokes in regular succession. Various diuretics were given, the digitalis was proposed, but he refused to take it. Crystals of tartar, the extractum lactucae virosae, nitrous aether etc., were tried without any benefit.

The symptoms of dropsy rapidly increasing, on the 9th of April, he took a draught of infusion of senna, tincture of jalap and Rochelle salts, which operated largely. On the 10th of April he was found in bed flushed, speechless, and hemiplegiac. How long he had been in that state could not be ascertained, as he had peremptorily ordered his servant not to remain in the chamber with him, and not to come to him in the morning till called. All attempts to relieve him were unavailing, his right side continued powerless, and his attempts to articulate were vain. The only peculiarity in the last period of his illness, which lasted eight or nine days was in the state of the respiration. For several days his breathing was irregular, it would entirely cease for a quarter of a minute, then it would become perceptible though very low, then by degrees it became heaving and quick, and then it would gradually cease again this

revolutıon ın the state of hıs breathıng occupıed about a mınute, durıng whıch there weıe about thıı ty acts of ıespıı atıon *

The Dıssectıon was made by Mr Cıampton, the Surgeon General, and wıtnessed by Mı John Moor and myself

There was nothıng remarkable ın the configuıatıon of the body but the great depth of the chest, the anasarcous swellıng of the ınferıor extremıtıes was consıdeıable

The scalp was bloodless The aıachnoıd membıane was slıghtly opaque, theıe was some fluıd between ıt and the pıa mateı, and the vascularıty of the latteı was ıncreased, moıe partıculaıly oveı the mıddle and posteııoı lobes of the cerebıum on the left sıde, wheıe, ın a laıge patch, ıt was thıckened and of a deep ıed colouı The bıaın was firm, ıts coıtıcal substance of a pale dıab colouı Theıe weıe between thıee and fouı ounces of fluıd ın the ventııcles

There weıe not moıe than two ounces of fluıd ın the peııcaıdıum The heart was about thıee tımes ıts natuıal sıze The lower paıt of the rıght ventrıcle was conveıted ınto a soft fatty substance, the uppeı part was remaıkably thın, and ıt gıadually degeneıated ınto thıs soft fatty substance The cavıty of the left ventrıcle was greatly enlarged The whole substance of the left ventııcle, wıth the exceptıon of the ınternal retıculated structure and caıneae columnae, was conveıted ınto fat The valves were sound The aorta was studded wıth steatomatous and earthy concretıons

*The same descrıptıon of breathıng was observed by me ın a relatıve of the subject of thıs case, who also dıed of a dısease of the heart, the exact natuıe of whıch howeveı I am ıgnorant of, not havıng been permıtted to examıne the body after death

1819

RENÉ THÉOPHILE HYACINTHE LAENNEC

THE INTRODUCTION OF THE STETHOSCOPE AND AUSCULTATION

RENÉ THÉOPHILE HYACINTHE LAËNNEC

(Courtesy Annals of Medical History)

RENÉ THÉOPHILE HYACINTHE LAENNEC

(1781-1826)

"I profess free medicine, I am not with the ancient nor with the modern, but seek the truth in each, and test everything by repeated trial."

—Laennec's credo from his doctoral thesis, after Webb

WHILE Auenbrugger, long past his prime, was leading a retired life in Vienna, a child of sickly appearance was born of a probably tuberculous mother at Quimper in Lower Brittany, one of the most beautiful districts in France. His name was René Théophile Hyacinthe Laennec.

The other immediate members of the family were a boy, Michel, who was born in 1782, and a girl, Marie, born in 1785. Another girl was born in 1786, but she lived only a few days and the death of the brave mother of this family occurred shortly afterward. The father was a lawyer and to judge by subsequent records, was not a very successful one. He also wrote poetry and that not too well.

After the death of the mother, the father, seemingly unable to accept the responsibilities of rearing his sons, sent the two boys to live with their paternal uncle, Michel, the rector of a parish in Elliant. This clergyman shortly afterward became one of the émigrés to England. And so in 1788, after spending about a year with Uncle Michel, the boys were sent to their other and more famous uncle, Guillaume-François Laennec, a former pupil of John Hunter and professor of medicine at the University of Nantes. Dr. Guillaume Laennec was interested in many things besides medicine. He enjoyed the humanities, was proficient in the Greek classics, derived pleasure from writing and was an effective speaker. René profited much from this association and indeed his physician uncle was more than a father to him.

For three years the two boys studied at L'Institution Tardivel, and in 1791, René was placed in the Collège d'Oratorie. There he studied religion, political science, orthography, grammar, geography, Latin prose and verse.

Contrasted to the peaceful life René was leading as an individual was the turmoil in which his country was embroiled. For by this time the French Revolution was in full sway. The new powers, Mirabeau, Danton, Robespierre, Marat and Carnot, were reshaping the destinies of millions. Even the son of sixty kings, now Citizen Louis Capet, was on his way to the guillotine. René, himself, must have felt the horror of this bloody revolution for he had seen several heads drop from the guillotine which was stationed in the square outside of his home in Nantes. His uncle was imprisoned for six weeks on suspicion of being out of sympathy with the contemporary government.

Despite the political confusion and chaos caused by the Revolution, the education of Laennec was not neglected. In 1793 he entered the National Institute. In 1795, encouraged by his uncle, Laennec began the study of medicine at the early age of fourteen and a half years. L'Hôtel Dieu at Nantes, where he began his study, was a large hospital, containing 400 beds. His uncle had charge of 100 beds, most of these were occupied by sailors suffering from tropical diseases. Besides medicine, Laennec studied botany and he also found time for the study of Greek at L'École Centrale.

THÉOPHILE LAËNNEC ON HIS ROUNDS IN THE NECKER HOSPITAL IN PARIS

From a painting by Chartrau

(Courtesy Roche Review)

After spending five years at Nantes, Laennec, at the insistence of his uncle, was sent by his father to Paris. There he immediately enrolled at L'École de Médecine, which at that time was championed by the great Jean Nicolas de Corvisart. Corvisart was the founder of French clinical medicine, and when Napoleon by chance became Corvisart's patient, the Emperor singled him out to be his personal physician. It was Corvisart, also, as we have shown, who caused Auenbrugger's discovery of percussion to be broadcast throughout the medical world. From all accounts, Laennec got along well with his famous teacher and received much genuine encouragement from the master on the theories that he advanced. It is interesting to note in this connection that at some time later, the aphorisms of Corvisart were published by Laennec, who had collected and preserved them during his residency in Paris.

Other famous teachers of Laennec were Marie François Xavier Bichat (1771-1802), whose work in physiology and medicine resulted in the founding of pathologic anatomy and scientific histology, and Baron Guillaume Dupuytren (1778-1835), whom Garrison described as "the ablest and best trained French surgeon of his time."[1] Dupuytren tolerated no rivals and consequently he and Laennec did not get along.

In 1802 Laennec observed, during necropsy on the body of a patient who had had cardiac disease, ossification of the mitral valves and dilatation of the ventricle. His report of the case constituted his first published work. Laennec's lecture in March, 1804, on tuberculosis, delivered shortly before his graduation, established the fact that phthisis was simply tuberculosis of the lungs. From that time onward the disease was called "pulmonary tuberculosis."

Laennec received his doctor's degree in June, 1804. His thesis was entitled "Propositions on the doctrines of Hippocrates in regard to the practice of medicine."

For five years after graduation Laennec busied himself lecturing on pathologic anatomy, doing what private practice he could, and contributing several articles to medical journals, and to the medical dictionaries and encyclopedias which were then in fashion. Many of his articles were published in the "Journal de Medecine" of which he was an editor from 1805 to 1808. It is apparent from some of his notes and editorials published therein that he was violently opposed to the nebulous theories of John Brown (1735-1788) and François J. V. Broussais (1772-1838). Brown's therapeutic ideas, according to Baas,[2] destroyed more people than did the French Revolution and the Napoleonic wars combined. Broussais thought that life depended on irritation and that disease owed its existence to localized irritation of the affected part. Broussais felt that nature had no healing power and therefore his therapy consisted of starving the patient and applying leeches over various parts of the body. Broussaism was such a popular doctrine that seven years after Laennec's death (1833) 41,500,000 leeches were imported into France to be used for the purposes of bleeding.

In 1812 Laennec was appointed physician to the Beaujon Hospital. During this time he especially interested himself in diseases of the chest and, of course, he employed percussion in his diagnosis as he had learned it from his master, Corvisart.

Laennec became associated with the Necker Hospital in 1816. It was at this time, also, that he developed the art of auscultation. The story of his discovery is admirably told in his own words in the introduction to the second part (diagnosis) of his famous book, "De l'auscultation mediate."

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[1]Garrison, F. H.: *An Introduction to the History of Medicine*. Ed. 4. Philadelphia, W. B. Saunders Company, 1929, p. 488.
[2]Quoted by Garrison, p. 315.

"In 1816, I was consulted by a young woman labouring under general symptoms of diseased heart, and in whose case percussion and the application of the hand were of little avail on account of the great degree of fatness The other method just mentioned being rendered inadmissible by the age and sex of the patient, I happened to recollect a simple and well-known fact in acoustics, and fancied at the same time, that it might be turned to some use on the present occasion The fact I allude to is the augmented impression of sound when conveyed through certain solid bodies, as when we hear the scratch of a pin at one end of a piece of wood, on applying our ear to the other Immediately, on this suggestion, I rolled a quire of paper into a sort of cylinder and applied one end of it to the region of the heart and the other to my ear, and was not a little surprised and pleased, to find that I could thereby perceive the action of the heart in a manner much more clear and distinct than I had ever been able to do by the immediate application of the ear From this moment I imagined that the circumstance might furnish means for enabling us to ascertain the character, not only of the action of the heart, but of every species of sound produced by the motion of all the thoracic viscera "

With the stethoscope René Laennec first heard the language of pathology Lesions within the thorax that for centuries had been inaudible now announced their presance At first, Laennec's discovery was treated with indifference by his immediate colleagues But although even his book met with a cold reception from these men, the first edition of 3500 copies was soon exhausted, and his deserved fame as a clinician and his pathologic work placed him on a firm footing His foreign colleagues greeted his discovery with much enthusiasm and soon physicians from all over Europe crowded into the Necker Hospital to hear his ideas on auscultation At last his own colleagues were convinced of the validity of Laennec's discovery Laennec was made professor of medicine in the Collège de France in 1822 and simultaneously became a member of the Academy of Medicine of France A year later he succeeded his teacher, Corvisart, in the College He also at that time was appointed physician to the Duchess of Berry In 1824 Laennec was made a knight of the Legion of Honor

That same year Laennec married a widow, Madame Argou It was a marriage of convenience, not of love The widow, who had been his housekeeper, was about forty-five years of age and ill health made her look older Laennec, himself, presented an emaciated appearance, suffering as he was from asthma

In 1826 Laennec published the second edition of his work on mediate auscultation This entailed considerable hard work and his health, which had never been robust, broke down In April of 1826, he contracted a severe cold This was accompanied by infection of the throat, high fever, and thoracic pains From that time onward his health became progressively worse He died on August 13, 1826

Except for his treatise on auscultation (1819), Laennec's most important contributions to medicine were produced during the beginning of his career They included his description of the pathologic appearance of peritonitis (1803) and a description of the capsule of connective tissue investing the liver (1803) In 1806 Laennec published the first accurate account of melanosis, and in 1812 he described an extraperitoneal type of hernia He was also the first to describe chronic diffuse interstitial hepatitis

The "Boston Medical and Surgical Journal" recorded in 1867 that 20,000 francs had been raised during that year for a monument to Laennec, the money having been raised chiefly in France, but in part by the Medico-Chirurgical Society of London and also by physicians in Scotland, Ireland, Prussia and Austria Modelled by Lequesne and cast by Ducel, the monument was exhibited at the Paris Exposition and was dedicated in May of 1868

A

TREATISE

ON THE

DISEASES OF THE CHEST.

IN WHICH THEY ARE DESCRIBED

ACCORDING TO THEIR

ANATOMICAL CHARACTERS.

AND THEIR

DIAGNOSIS

ESTABLISHED ON A NEW PRINCIPLE

BY MEANS OF

ACOUSTICK INSTRUMENTS.

With Plates.

TRANSLATED FROM THE FRENCH OF

R. T. H. LAENNEC, M. D.

WITH

A PREFACE AND NOTES,

BY JOHN FORBES, M. D.

PHYSICIAN TO THE PENZANCE DISPENSARY, SECRETARY OF THE ROYAL GEOLOGICAL SOCIETY OF CORNWALL, &c. &c.

FIRST AMERICAN EDITION.

PHILADELPHIA.

JAMES WEBSTER, 24 S. EIGHTH STREET.

William Brown, Printer.

1823

TREATISE ON MEDIATE AUSCULTATION*

PREFACE

I BEGAN, three years since, the researches of which I now publish the results. Although these have not reached the degree of perfection which longer experience would have conferred on them, I have thought it advisable for many reasons, to communicate them to the public. Among those reasons I may mention—the incorrect accounts of my discoveries that have found their way into the journals of the day, the favourable report of the Academy of Sciences,† and the hope and conviction that the mode of exploration detailed in this work will be confirmed and extended by other observers.

It will be found that of the facts narrated in my treatise, I have given some as certain, others as doubtful, and a few merely as problematical. Of the first class, if future experience should invalidate any, I may venture to believe that the number will be few, and I am even convinced that the greater part of those which I have stated as doubtful, will be found by further observation to be constant and certain.

In respect of the pathological details, which constitute so large a portion of the work, I think it necessary to make a few observations. The great attention that has been paid to morbid anatomy, since the commencement of the present century, throughout Europe, and more especially in Paris, has been productive of many improvements and discoveries which are but imperfectly known, and, indeed, many of which have not at all been communicated to the public, at least by their discoverers. On this account, the present state of our written knowledge is obviously behind our actual knowledge, and if, in the present work, I had contented myself with merely describing the signs of the organic lesions, without describing the lesions themselves, I should have often

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*Laennec, R. T. H. *Traité de l'auscultation médiate.* The first French edition was published in 1819. We are reprinting from the first American edition, published in 1823.—F. A. W., 1940.

†Extract from the Report of the Academy of Sciences (drawn up by M. Percy, and signed by him and MM. Portal and Pelletan, 29th June 1818) on a Memoir of M. Laennec respecting the use of Auscultation, more particularly in Phthisis Pulmonalis.

"The Cylinder applied to the chest of a healthy person who sings or speaks, produces a sort of vibration which is more distinct in some places than others. But when there exists an ulcer in the lungs, the patient's voice then, instead of being heard in the usual manner by the exposed ear, reaches the other entirely through the tube of the instrument. We have ourselves verified this fact on several consumptive patients, it appeared to us striking, and well fitted for furnishing a certain and easy sign of certain morbid conditions of the lung, which, in the present state of medicine, can only be suspected to exist.

"We have also examined by means of the cylinders, the respiration in different parts of the chest of a healthy person, and found it very distinctly audible in every point of this cavity which corresponded with the lungs. We have also found that the motions of the heart were equally perceptible, and it has consequently, appeared to us that the assertions of the author, of the possibility of obtaining, through these two kinds of auscultation, certain signs of the several diseases of the heart and lungs, were at least, extremely probable."

run the risk of being not understood at all, or (what is worse) of being misunderstood I have, therefore, felt that the only means left of escaping this danger, was to give an anatomical description of all the diseases of which I have noticed the symptoms In fulfilling this task I have endeavoured to render my descriptions concise, yet, at the same time, sufficiently exact and complete to characterise the objects

Another motive has contributed to strengthen this resolution —viz the conviction of the practical utility of my mode of diagnosis, and the belief that the surest way of procuring its more general adoption was to associate the exposition of its principles with a description of the diseases which it indicates, more exact than any that yet exists

Many reasons have induced me to prefer the anatomical to the more symptomatical description of diseases The former method has the advantage of brevity, perspicuity and certainty It is, for example, much easier to describe tubercles and detail the signs of these, than to define the disease by the external symptoms only, and to arrange its varieties according to their *causes* Emphysema of the lungs consists in an alteration of parts which can be described in a few words, and of which the signs can be easily recognised, while in studying asthma, according to the method of Sauvages, we shall require to write a volume on generalities before we can arrive at anything positive

It will, perhaps, be objected that the anatomical method has the disadvantage of founding its species on distinctions, the chief characters of which can only be obtained after death but this objection scarcely merits refutation We might as well say that it is useless for surgeons to make any distinction between dislocation of the femur, and fracture of its neck, or that it is useless to separate bronchitis from peripneumony

The morbid alteration in the affected organ is, unquestionably, the least variable and most positive of the phenomena of local disease, it is on the nature and extent of this alteration that the danger and curability of diseases always depend, and it is this, consequently, that ought to be considered as characterising them On the contrary, the derangement of functions which accompanies these alterations is extremely variable it is often the same under circumstances entirely different, consequently, it can rarely serve to discriminate different diseases

Besides, it is a mistake to consider the recognition of nosological species, founded on the data of morbid anatomy, as impracticable before death on the contrary, they are often more readily recognised during life, and certainly present to the mind something much clearer and more positive, than any nosological distinction founded on the symptoms merely Peritonitis, for example, is assuredly a disease easily distinguished during life, and out of twenty medical men acquainted with morbid anatomy called to see a case of it not one will make a mistake

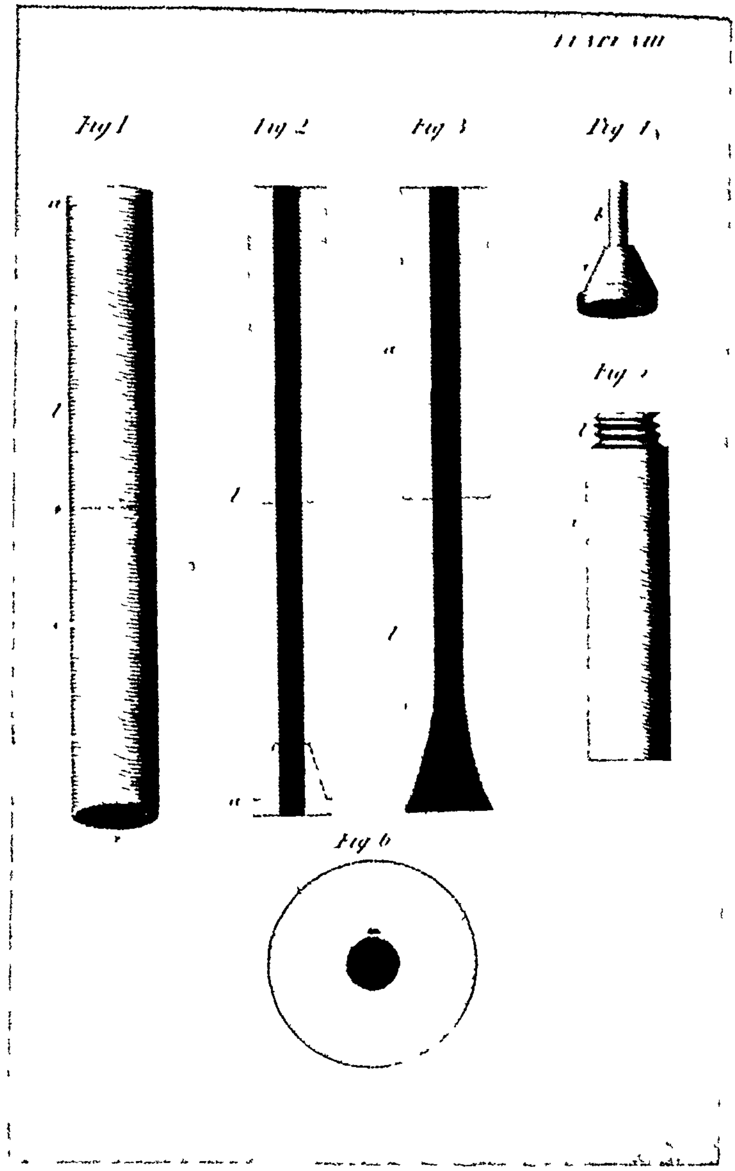

PLATE VIII

Fig 1 The Stethoscope or Cylinder, reduced to one third its actual dimensions *a* Stopper *b* The lower end *c* The upper half *d* The auricular or upper extremity

Fig 2 Longitudinal section of the same *a* The stopper *b* Point of union of the two parts *c* The upper half

Fig 3 The same section, with the stopper removed

Fig 4 The stopper *a* The body of it, formed of the same wood as the rest of *the instrument b Small brass tube traversing the stopper, for fixing it in the tube of* the stethoscope

Fig 5 Upper half of the stethoscope *a* Body of it *b* Screw (in the wood) for fixing the two portions together

Fig 6 Actual diameter of the stethoscope

N B Any turner will be able to make the instrument, from the above description —*Trans*

concerning its nature or name But will this be the case with those who are accustomed to see in diseases nothing but symptoms? Of the twenty shall we not find one considering the affection as *ileus,* another as *hepatic colic,* a third as *puerperal fever*, and so on? The same thing may be said of peripneumony, nephritis, hepatitis etc , and I hope that the work now submitted to the public will enable us to say the same thing of most of the diseases of the lungs, pleura and heart

Morbid anatomy must, then, I think, be considered as the surest guide of the physician, as well to the diagnosis as to the cure of diseases But it must not be forgotten that it has also its obscure points It is, no doubt, an easy matter to distinguish striking changes of structure, but there are many slighter alterations, among which it is difficult to ascertain what is healthy and what diseased, what cause and what effect, and, lastly, whether the appearances are truly the effect of disease, or merely an accident of assimilation, or circulation, that has taken place in articulo mortis, or even after death In these cases we must content ourselves with what is clear and distinct, never forgetting in practice the principle of Hoffman—*Nunquam aliquid magni facias ex mera conjectura aut hypothesi,* and sedulously guarding against the error of believing that the mere knowledge of the seat and nature of the disease can justify our neglecting its individual character, as influenced by external circumstances or personal idiosyncrasy

From the foregoing observations it will be seen that this work is not, like that of Auenbrugger, a simple exposition of new means of diagnosis Neither can it be considered as a monography of the diseases of the chest,—since I have taken little notice of the ordinary and more general symptoms of the diseases, and have not at all touched upon their treatment

In the construction of my treatise I have quoted but two authors The chief object of my researches was, in a great measure, new, and for the facts already known respecting the diseases of the lungs and heart, I have thought it unnecessary to go beyond the works of M Corvisart and Bayle* If I have occasionally differed from these distinguished authors, I trust no one will misinterpret my motives No one can be more sensible of their merits, both as men and Physicians, than myself At the very time I question their opinions, I most willingly confess my great obligations to them It is much easier to improve a field already cultivated, than to reclaim a wild and barren soil In respect of the works of M Corvisart more particularly it is to be regretted that those of them published by others are far from giving a just idea of the

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**Essai sur les maladies et les lesions organiques du coeur, etc* par J N Corvisart Translated by Hebb London Underwood 1813

Nouvelle methode pour reconnoitre les maladies internes de la poitrine par la percussion de cette cavite par Auenbrugger ouvrage traduit du latin et commenté par J N Corvisart Paris 1808

Recherches sur la Phthisie pulmonaire par G L Bayle Paris 1810 Translated by Barrow 1815

author's merits. The uncertainty of the signs of diseases, and the vagueness of description in these, appears peculiarly striking to those who, like myself, were his pupils, and habitual witnesses of the boldness and precision of his diagnostics. This defect, no doubt, partly depends on the incommunicable tact of the physician, which forms so great a part of the art, and which M. Corvisart possessed in the highest degree.

I have hopes that the advantages of my method of diagnosis may be extended, in some degree, to veterinary medicine. Many reasons, however, exist, why this art must derive inferior benefit from it. Among these I may mention the absence of the voice,—the comparative inaccessibility of the region of the heart, and, lastly, the great indistinctness of respiration in the horse, and probably all herbivorous animals. In cases of disease, however, the respiration will be more audible in the sound portions of the lungs, as I found in a case of peripneumony in a cow, which I recognised during the animal's life, as easily as in the human subject. In the dog, and cat, and probably in all carnivorous animals, the sound of respiration is as distinct as in man. Notwithstanding these difficulties, I have no doubt that further experience will prove the utility of mediate auscultation in the disease of animals, especially, if combined with percussion of the chest.*

OF THE ACTION OF THE HEART IN GENERAL, IN HEALTH AND DISEASE†

Before entering upon the diagnostic signs furnished by the stethoscope in particular diseases of the heart, it will be necessary to examine the general results afforded by it, as well in the sound as diseased state of that organ. I shall do this under four principal heads, viz. 1st, the extent of the heart's action, as ascertained by the cylinder; 2nd, the shock or impulse communicated; 3rd, the nature and intensity of the sound; and 4th, the rythm of its actions.

I. Of the Extent of the Pulsation of the Heart

This must be considered in two points of view:—first, the sensation conveyed by the instrument when applied to the region of the heart; and, secondly, the parts of the chest (other than this region) in which its action can be perceived.

*The author further suggests the probable utility of the stethoscope in the instruction of the deaf and dumb, by applying one end of it to the trachea of the speaker and the other to the ear of the pupil;—but surely this must be fanciful—or at least of inferior value to other means.—*Trans.*

†Laennec's work in the translation by John Forbes is composed of three books, Book First, Book Second and Book Third. We have reprinted portions of Book Third which deals first, with diseases of the heart and second with diagnosis of diseases of the heart. Excerpts from these two parts of Book Third are *not* reprinted in the sequence in which they appear in Forbes' translation. To aid the reader in distinguishing the two parts of Book Third, a star has been appended to the titles of all paragraphs taken from the first part of Book Third (on diseases of the heart). Paragraphs *not* marked with a star have been taken from the second part of Book Third (on diagnosis of diseases of the heart).—F. A. W., 1940.

1. In the natural condition of the organ, the heart, examined between the cartilages of the fifth and sixth ribs, and at the lower end of the sternum, communicates, by its motions, a sensation as if it corresponded evidently with a small point of the thoracic parietes, not larger than that occupied by the end of the stethoscope. Sometimes, it appears as if it were placed deep in the mediastinal cavity, leaving a vacant space between it and the sternum: in this case its movements, even when pretty energetic, appear to communicate no vibratory impulse to the neighbouring parts. In other cases, again, the heart seems entirely to fill the cavity of the mediastinum, and to extend much beyond the point on which the instrument rests, and, in this case, its contractions, even when slow and noiseless, seem to elevate, to a considerable extent, the thoracic parietes before them, and to displace the adjacent viscera within. This difference of sensation seems, in a word, to convey the impression of the action of a smaller or a larger heart, and, generally speaking, this indication is sufficiently correct, when the organ is examined in the state of quietude which results simply from repose of body.

2. The second point is of more practical importance. In a healthy person, of moderate fulness, and whose heart is well proportioned, the pulsation of this organ is only perceived in the cardiac region, that is, in the space comprised between the cartilages of the fifth and seventh ribs, and under the lower end of the sternum. The motions of the left cavities of the heart are chiefly perceptible in the former position, those of the right cavities in the latter. This is so much the case, that, in disease of one side of the heart only, the pulsation in these two situations gives quite different results. When the sternum is short, the pulsations extend to the epigastrium. In very fat subjects, the pulsation of whose hearts is quite imperceptible to the mere touch, the space in which it can be detected by the cylinder is sometimes not more than an inch square. In thin persons, in the narrow-chested, and, also, in children, the pulsation is more extended, being perceptible over the lower third, or even three-fourths, of the sternum, and sometimes even over the whole of this bone, also at the superior part of the left side, as high as the clavicle, and sometimes, though feebly, under the right clavicle.

When the pulsations are confined to the places above mentioned, in subjects of the kind noticed, and when they are much weaker below the clavicles than in the region of the heart, we may conclude that this viscus is well proportioned.

When the pulsations of the heart become more extended, they are heard successively in the following places:—1st. the whole left side of the chest, from the axilla to the stomach. 2nd, the whole of the right sides, 3rd, the posterior part of the left side of chest: and 4th. the posterior part of the right side. This last is rare. In these cases the intensity of the sound is progressively less in the succession mentioned. This succession has ap-

peared to be constant, and may be taken as an index of the extent of pulsation For instance, if this be perceptible on the right side, we may be assured that it will be equally so over the whole sternum, under both clavicles, and over the left side, but we are not sure that it will be so on the back But if it be perceptible on the back on the right side, we may calculate on its being still more audible in every other part of the chest

Several circumstances unconnected with the state of the heart may derange the order above mentioned, and augment the extent of the pulsation This latter effect is produced by a hepatized or compressed lung, and also by a part containing tuberculous excavations In every case the heart gives two distinct pulsations for one beat of the arterial pulse In my examinations of several hundred individuals, I have only met with one in whom the pulsation of the subclavian arteries could be heard by the stethoscope, and I may state it is an almost universal fact, that neither the pulsation of this artery, nor of the aorta, can be mistaken for that of the heart

When the pulsation of the heart is heard over a greater extent than what is above stated to be the range of a well proportioned organ, the individual rarely enjoys good health If he has not formal dyspnoea, he has, at least, shorter breath than usual, is put more easily out of breath, and is more subject to palpitation This state, however, which is that of many asthmatics, may remain stationary many years, and does not always prevent the attainment of an advanced age

With regard to the relation between the state of the heart and the extent of its pulsation, I think it may be taken as a general fact, that the extent of pulsation is in the direct ratio of the thinness and weakness of the heart, and consequently, inversely as its thickness and strength The size of the organ must also be considered as affecting the extent of its pulsation

In explanation of what has been just stated, we may presume, when the pulsation extends over all the places above mentioned, that the heart is increased beyond the natural size, and that this increase is owing to the dilatation of one or both ventricles This presumption will be strengthened, if the pulsation is as great under the clavicles or in the axilla, as in the region of the heart If the pulsation is perceived neither in the back nor right side, but only in the other points mentioned, and if its intensity is nearly equal in all these, we may conclude that the ventricles are moderately dilated, and that the parietes of the heart are naturally thin On the contrary, when there is very strong pulsation in the region of the heart, and none or very little under the clavicle, we may be assured (if the patient has other general symptoms of diseased heart) that the disease is hypertrophia of the ventricles If the patient has never experienced any marked disorder of the circulatory organs, we may be certain that the parietes of the left ventricle are much thickened, though still not sufficient to constitute disease

Generally speaking, then, it may be taken for granted that a great extent of pulsation is a mark of thin parietes of the heart, more particularly of the ventricles, and that a confined range of pulsation coincides with an increased thickness of these. Some accidental causes may augment for a time the extent of the heart's pulsation, such as nervous agitation, fever, palpitation, haemoptysis, and, in general, whatever increases the frequency of the pulse.

II. Of the Impulse Communicated to the Ear by the Action of the Heart.

In investigating this we must be careful not to confound with the action of the heart, the rise of the thoracic parietes during inspiration. This caution is more particularly necessary when the respiration is very short and frequent.

The degree of impulse communicated by the cylinder to the ear, is, in general, inversely as the extent of the pulsation of the heart, and directly as the thickness of the walls of the ventricles. In a person whose organs of circulation are well proportioned, this impulse is very little perceptible, often quite imperceptible, especially if the individual is rather fat. When the parietes of the heart are unnaturally thick, the impulse is usually so great as very sensibly to elevate the head of the observer, and sometimes to give a disagreeable shock to the ear. The more intense the hypertrophia, the longer time the impulse is perceptible. When the disease exists in a high degree, we feel as if the heart, in dilating, first comes in contact with the thoracic parietes in one point only, and then with its whole surface, and that it contracts and falls back all at once. The impulse of the heart is only felt during the systole of the ventricles, or if, in some rare cases, an analogous phenomenon accompanies the contraction of the auricles, this is easily distinguished from the former. In fact, when the systole of the auricles is attended by any sensible action, this is perceived to have its seat much deeper, and most commonly it consists merely of a sort of vibration. In any case, it is very little marked as compared with the sensation produced by the contraction of the ventricles, when these are of a good degree of thickness.

When the parietes of the heart are thinner than usual, no impulse is communicated, even when the pulsation is the greatest, and, in this case, the alternate contraction of its cavities is only distinguished by the sound these produce. A strong impulse, therefore, must be regarded as the chief sign of hypertrophia, and the absence of all impulse as the characteristic of dilatation of the heart. The correctness and constancy of this result have been confirmed to me by many examples.

The impulse of the heart's action is usually perceptible only over the region of the heart or, at most, over the inferior half of the sternum. When very great it extends to the epigastrium in cases where the sternum is short. In simple hypertrophia, it is usually perceived in no other part,

but when this is conjoined with a certain degree of dilatation, it is sometimes distinctly perceived under the clavicles, and in the right side of the chest The impulse of the heart's action is, of course, diminished by whatever debilitates the general strength of the system

III Of the Sound Produced by the Action of the Heart

The alternate contraction of the different parts of the heart produces a peculiar sound, of which the individual is himself sensible during palpitation and in fever In certain states of disease it can be heard at some distance from the patient, but this is a very rare case The sound is the only phenomenon usually observable in any other part of the chest beside the precordial, the impulse of its action being confined, as already observed, to that part

The sound produced by the action of the heart is great in proportion as the parietes of the ventricles are thin and their impulse feeble consequently, it cannot be attributed to the percussion of this organ against the side In a moderate degree of hypertrophia, the contraction of the ventricles yields only a dull sound, like the murmur of inspiration, and the auricle, in like manner, a much less noise than in the natural state In a high degree of hypertrophia, the contraction of the ventricles produces merely a shock without any sound, and the sound of the auricles is scarcely audible On the other hand, when the ventricular parietes are thin, the noise produced by their contraction is clear and loud, approaching to that of the auricles, and if there be a marked dilatation of the ventricles, the sound becomes very similar, and almost as strong as that of the auricles

In the state of health the sound of the contractions of the heart is nowhere heard so strongly as in the region of the heart In certain states of disease it may be heard more distinctly in other places

The softening of the substance of the heart deadens the sound of its contractions, as does also any impediment of the circulation, whether caused by too much blood, or by an obstacle in the auriculo-ventricular orifices This latter state, further, gives rise to a dull rustling sound, very like the noise of bellows, or (when stronger) like that produced by the action of a file on wood The particular orifice affected is, in this case, indicated by the place and time in which the sound is observed When the orifice is on the left side, we can sometimes feel with the hand a sort of vibratory sensation like that produced by the purring of a cat In this case, the noise produced by the contraction of the cavity having the obstructed orifice is not only duller, but much more prolonged than in the natural state

IV Of the Rythm of the Pulsations of the Heart

By rythm I understand the order of the contractions of different parts of the heart, and their relative duration and succession, as detected by the cylinder Before entering on this subject I think it necessary to notice

the relative proportions of the heart to the body of the individual, and of the different parts of the heart to each other, in a state of health, and in a well proportioned subject

The heart, including the auricles, ought to be of a size equal to the closed hand of the subject, or only a little less or greater than it The walls of the left ventricle ought to be of a thickness somewhat more than double that of the right The texture of the left ventricle, firmer and more compact than that of the muscles, ought to keep it from collapsing when laid open The right ventricle ought to be a little larger than the left, with columnae carneae of greater size, and ought to collapse on being cut into In a heart so proportioned, the alternate contractions of the ventricles and auricles, as examined by the cylinder, and the pulse as examined by the finger, afford the following results —

At the moment of the arterial pulse, the ear is slightly elevated by an isochronous motion of the heart, which is accompanied by a somewhat dull, though distinct sound This is the contraction of the ventricles Immediately after, and without any interval, a noise resembling that of a valve, or a whip, or the lapping of a dog, announces the contraction of the auricle (I make use of these trivial expressions because they appear to me to express better than any description, the nature of the sound in question) This noise is accompanied by no motion perceptible by the ear, and is separated by no interval of repose from the duller sound and motion indicative of the contraction of the ventricles, which it seems, as it were, to interrupt abruptly The duration of this sound, and consequently the period of contraction of the auricles, is less than that of the ventricles,—an incontestible fact of which Haller entertained doubts Immediately after the systole of the auricles there is a very short, yet well-marked interval of repose, subsequently to which we feel the ventricles swell anew, with the dull sound and gradual progression which characterise their action, then follows the quick and sonorous contraction of the auricles, and again the renewed but momentary immobility of the heart This state of quietude after the contraction of the auricles does not appear to have been known to Haller as a natural condition The relative duration of the contractions of the auricles and ventricles, appears to me to be as follows Dividing the whole into four parts, a fourth (or third) belongs to the systole of the auricles, a fourth (or somewhat less) to the state of quiescence, and two-fourths to the systole of the ventricles—These observations are most conveniently made when the pulse is slow

From the foregoing observations it appears that the heart, far from being in a state of constant action, as is usually supposed, presents alternations of action and respose, the sum of which does not differ from those of many other muscles, more especially the diaphragm and intercostal muscles From the proportions above stated it follows that in twenty-four hours

the ventricles have twelve, and the auricles eighteen hours of quiescence. In persons whose pulse is habitually below 50, the repose of the ventricles is more than sixteen hours in the four-and-twenty.

Hypertrophia of the ventricles, when in a moderate degree, presents, in some respects, an exaggeration of the natural rythm of the heart's action. The contraction of the ventricles becomes less noisy, and more readily distinguishable from that of the auricles. After the latter, the interval of quiescence is well-marked and contrasts very sensibly with the sound that precedes, and the motion which follows it. But in hypertrophia carried to a very high degree the rythm of the heart is singularly changed.

In this case, the contraction of the ventricles is greatly prolonged. This at first is perceived as a profound and obscure motion, which gradually augments, elevates the applied ear, and then terminates in producing the impulse or shock. This contraction is unaccompanied by any noise, or, if this exists, it is merely a sort of murmur like that of respiration.

The contraction of the auricles is extremely short, and almost, or altogether, without sound, and in some cases the systole of the ventricles seems scarcely over before they begin to swell afresh.

In extreme cases there is no sound distinguishable but the murmur above mentioned, and we merely recognise an elevation of the heart corresponding to each beat of the pulse. In these cases the increased brevity of the auricular contraction is not the consequence of their diminished contractibility merely, but, also, of their contraction commencing before that of the ventricles has entirely ceased.

When the walls of the left ventricle are naturally thin, or have become so from dilatation, the rythm of the heart's actions is quite different. In this case, the interval of repose after the contraction of the auricles is no longer perceptible. The contraction of the ventricles is more sonorous, more resembling that of the auricles, and more approaching the latter in duration. In this condition of the heart, there is as already observed, a less degree of impulse during the contraction of the ventricles, and a greater extent in the pulsation of the heart. This condition of the organ of circulation is congenital in many cases. It does not necessarily abridge life, but is usually conjoined with a delicate constitution.

Actual dilatation of the heart produces merely an increase of all the characters which indicate a heart with thin parietes. The contraction of the ventricles becomes as short and noisy as that of the auricles, the pulse, consequently, becomes very frequent, and the isochronism of the arterial pulse and the contraction of the ventricles becomes quite indistinguishable. In addition to these signs we must add—the absence of any sensible impulse, the extension of the heart's pulsation over the whole or greater part of the chest, and the existence of this in as great force under the

clavicles and the axilla as in the region of the heart itself This last character, particularly, may be regarded as pathognomonic, if the patient is not phthisical and pectoriloquous in the places mentioned

V Of Palpitation of the Heart

By palpitation of the heart is meant, in the common language of medicine, every beating of the heart which is sensible and unpleasant to the individual, and, at the same time, more frequent than natural When this affection is studied by the aid of the cylinder, we find that there are many varieties of it, all of which appear to have merely this one character in common, viz that the individual is sensible of the heart's action Frequently, also, the patient *hears* the pulsation, especially when in the horizontal posture In the upright position, the contraction of the ventricles only is heard, while, when lying on the side, the individual is sensible of a pulsation of his ear double that of the pulse, viz the alternate contraction of both the ventricles and auricles In many cases there is merely an increased frequency of pulsation, although the patient imagines, from his sensations, that there is also great increase of force This species of palpitation is most common in dilatation of the ventricles, and lasts the longest of any I have known it continued eight days, the pulse remaining, through the whole of this time, extremely small and weak, and between 160 and 180

Another variety consists in an increase both of frequency and force of pulsation This is what arises in healthy persons from great exertion or from moral causes, it also accompanies slight degrees of hypertrophia In simple hypertrophia in a high degree, the ventricles are found to contract with great force, and to elevate the thoracic parietes in an extent and to a height much greater than natural The noise, however, produced by their contraction is much duller and more indistinct than usual, the extent of thorax over which the pulsation is perceptible is not increased, and, notwithstanding the increase of the heart's power to double or triple its ordinary force, the pulse is, almost always, two or three times more feeble and smaller than in the natural condition of the circulation In hypertrophia with dilatation, the impulse, noise, and extent of the heart's action, are usually equally increased

VI Of Irregularity of the Heart's Action

Irregularity in the pulsation of the heart may exist without palpitation In old persons this is often met with without any perceptible alteration of the general health The irregularity which occurs in palpitation consists usually in mere variations in the frequency of the heart's pulsation Sometimes this variation is almost constantly recurring, at other times it is at longer intervals, and consists only of a few contractions longer or shorter than the rest These irregularities occur most frequently in cases of dilatation

In hypertrophia, and during the existence of palpitation, the contractions of the ventricles are so quick, and so much prolonged, that those of the auricles cannot be perceived. It sometimes, though very rarely, happens during palpitation, that each contraction of the ventricles is followed by several successive contractions of the auricles, so quick as only to equal in point of time one ordinary contraction. Sometimes these contractions are two or four, but most frequently three.

VII. Intermission of the Pulsation of the Heart

By intermission, we usually understand a sudden and momentary suspension of the pulse, during which the artery is no longer perceptible beneath the finger. The duration of the intermission is very variable, and may serve to divide this affection into well-marked varieties. Sometimes the intermission is shorter than one arterial pulsation, sometimes it is equal, and sometimes it is longer.

The first kind of intermission is the most common, it is frequent in old age, even during health. At other periods of life, it is only observed in certain diseased states of the heart, particularly hypertrophia. By means of the stethoscope we ascertain that this species of intermission always succeeds the contraction of the auricles. It, therefore, only differs from the natural quiescence after this contraction, in the irregularity of its recurrence. The duration and recurrence of this species of suspension of the heart's action are very variable. This, the real intermission, must be distinguished from the false intermission, already noticed, produced by the variation of the duration and strength of the heart's contractions. This can easily be done by the cylinder. The species of intermission which consists in the absence of one complete pulsation, returning sometimes with an exact periodicity at longer or shorter intervals, constitutes the sign deemed by Solano indicative of the approach of critical diarrhoea. The third variety is accompanied by a state of fulness of the artery during its continuance.

Many considerations, some of which have been stated, prove that the mere examination of the pulse is insufficient to inform us of the true state of the circulation, and must often lead us into error.—To notice only the indications afforded by it as to blood-letting, to prognosis in all diseases, and to diagnosis in several.—

We have seen, that, in peripneumony and pleurisy, the absence of fever and a perfectly natural state of the pulse, frequently accompany an incurable disease. In diseases of the heart, the pulse is often feeble, sometimes even almost imperceptible, although the heart's contraction, that especially of the left ventricle, is much more energetic than natural. In apoplexy, on the contrary, we often meet with a very strong pulse in persons in whom the impulse of the heart's action is scarcely observable. These two opposite facts may easily be verified by the use of the cylinder. I have

myself done so, perhaps more than a thousand times, within the last three years. They appear quite inexplicable, unless we admit the arteries to possess a power of action independent of that of the heart.

It would seem to be proved, also, by many other facts, that the different systems subservient to the circulation, although necessarily and reciprocally dependent, have still, in other respects, a particular or individual existence, which, in certain states of the disease and in certain individuals, is more marked and isolated than in ordinary cases and circumstances. This view of the case is supported by the observations of practitioners, in all ages, of the different effects of bleeding, according as it is general or local, venous or arterial, depletive or derivative. The same is shown by the great benefit of a natural hemorrhage of a few ounces only, and the inefficacy of copious venesection in the same case; and by the trifling degree of exhaustion produced sometimes by very profuse hemorrhage, compared with the great collapse occasioned by the bleeding of a few leeches in the same person. These facts prove, I think, that the capillary circulation is in some sort independent of the general. The influence of the latter on the former seems very inconsiderable indeed in certain hemorrhages from the uterus, bowels, nose or lungs, which are found to be very little affected by the most copious venesection.

The mere state of the pulse, then, is far from indicating the state of the circulation in general; it does not even certainly indicate its condition in the whole heart, as it merely corresponds with the contraction of the left ventricle, which may be regular at the time when that of the auricles and right ventricle is irregular. In like manner, the state of the pulse fails to be a sure guide as to the expediency of blood-letting. Every one knows that in certain cases, for instance in apoplexy, peripneumony, pleurisy, and inflammatory affections of the abdomen, the weakness and smallness of the pulse do not always contra-indicate venesection; on the contrary, that the artery, in such cases, recovers its force and fulness after the loss of blood. The recognition of this kind of pulse (*fictitie debilis*) is one of the most important and difficult points in the treatment of the acute diseases, as an error in respect of it may be fatal. In cases of this sort, the stethoscope affords a rule much surer than the pulse. Whenever the contraction of the ventricles is energetic, we may bleed without fear,—the pulse will rise; but if the contractions of the heart are feeble, although the pulse still retains a certain degree of strength, we must be cautious respecting the employment of venesection. When the pulse is very strong, and the contractions of the heart moderately strong (as is frequently the case in apoplexy), we may still bleed with advantage as long as there is not a marked diminution in the noise and impulse of the heart's actions. But when both the pulse and the heart are feeble, we must not open a vein, whatever be the *name* of the seat of the disease, as such practice must infallibly destroy the few resources still left to nature. The most we can

do, in such a case, if there be any local congestion, is, to try, by the application of a few leeches, if the patient can bear the subtraction of blood from the capillaries

The certainty and facility with which the cylinder indicates the propriety of blood-letting in such cases as those above mentioned, (which have been hitherto considered among the most difficult in practical medicine,) appears to me to be the greatest advantage to be derived from the employment of this instrument

After what has been said, and after its general uncertainty avowed by the most experienced practitioners, it may seem surprising that the practice of feeling the pulse has been so generally followed in all ages The reason of this practice is, however, sufficiently obvious it is of easy performance, and gives little inconvenience either to the physician or patient, the cleverest, it is true, can derive from it but a few indications and uncertain conjectures, but the most ignorant can, without exposing themselves, deduce from it all sorts of indications Its very uncertainty gives it a preference with persons of inferior qualifications, over means quite certain in their nature, and which enable the non-professional observer to judge of the skill of the physician by the correctness of his diagnosis and prognosis

The facts above stated relative to the discordance existing between the pulsation of the heart and of the arteries,—more especially as to strength, are contrary to the more general opinion of modern physiologists, who consider the *action of the arteries as entirely dependent on that of the heart* Bichat himself has fallen into this error *

OF SYMPTOMS COMMON TO ALL THE DISEASES OF THE HEART

These are an habitually short and difficult respiration, palpitations and oppression constantly produced by the action of ascending, by quick walking, by emotions of mind,—or without any perceptible cause, frightful dreams, and sleep frequently disturbed by sudden starts, a cachectic paleness and a tendency to anasarca which, indeed, comes on after the disease has persisted some time To these symptoms is frequently added the *angina pectoris*,—a nervous affection characterised by a sense of oppression, constriction and oppression in the region of the heart, and a pain or numbness of the arm, more commonly of the left, sometimes of both at once When the disease has reached a high degree it is recognised at a single glance The patient, unable to bear the horizontal posture, remains night and day seated in his bed, with the face more or less swollen, sometimes very pale, but more commonly of a deep violet tint, either over the whole or only on the cheeks The lips are swollen and prominent, of a deeper violet than the rest of the face, or of this hue when it is quite pale The whole body is more or less anasarcous The congestion and lentor of

*Anat Gener 1re part tom II page 371

the capillary circulation are further shown by affections of the internal organs, for instance—haemoptysis, pains of the stomach, vomiting, apoplexy (which frequently terminates such affections), and most of all, dyspnoea, which last symptom has been the cause of confounding such diseases (with many others) under the name of *Asthma*. Emphysema of the lungs likewise bears much resemblance to some varieties of disease of the heart, but the following marks will distinguish them from each other.

In disease of the heart, the patient, although with the respiration habitually short, does not usually experience the feeling of oppression and dyspnoea, except when walking rather quick, or using much exertion, or, more particularly, when ascending an elevation.

On the other hand, the individuals affected with emphysema, become oppressed on the breath when they are quite still and these attacks recur without any known cause, or from a slight change of the weather. Moderate exercise seems often to relieve them, if the disease has not reached a great degree of intensity.

In diseases of the heart the general circulation is not always so much affected as the capillary. Sometimes the pulse is almost natural, but is often irregular.—At all events, it is evident that none of the general symptoms already mentioned suffice to characterise disease of the heart, and that for a certain diagnosis we must recur to mediate auscultation. It is necessary here to remark that the study of the physiological conditions of the heart, by means of the cylinder, requires much more time and application than that of the voice and respiration. In hospital practice, also, owing to our general ignorance of the anterior history of patients, we are liable to be led into error by its use, without proper care. For example, we may, in some cases, consider a patient as labouring under hypertrophia or dilatation of the heart, when he is merely affected with nervous palpitations. Another, and more insidious cause of mistake, arises in diseases which diminish the extent of respiration, for instance, peripneumony, emphysema, and more particularly chronic pleurisy. In cases of this kind I have sometimes found the heart enormously dilated and thickened after death, although, during life, its contractions had been perfectly natural in respect of sound, impulse and rythm. It would seem as if the diminished capacity of the lungs produced a diminished action of the heart.

*OF HYPERTROPHIA, OR SIMPLE ENLARGEMENT OF THE HEART**

By Hypertrophia I mean simple increase of the muscular substance of the heart, without a proportionate dilatation of its cavities, or even with a diminution of these. This affection is by no means common, and appears to have escaped the notice of M. Corvisart, as, through his whole work, he

*Book Third. Chapter 1. Section First. See note on page 332.—F. A. W., 1940.

seems to consider enlargement of the parietes of the heart, as being uniformly accompanied by a proportionate dilatation of the cavities of that organ

This enlargement of the heart is always attended by a considerable increase of its consistence, except when conjointed with another affection of this organ, to be noticed presently, viz *softening of the heart*

Hypertrophia may exist in one or both ventricles, with or without a similar affection of the auricles Most commonly the auricles are not affected, but occasionally they are so, while the ventricles are sound

When affecting the left ventricle, I have seen its parietes more than an inch thick at the base, that is, double that of its sound state Commonly, this morbid thickening diminishes insensibly from the base to the apex of the ventricle, where it is scarcely perceptible, sometimes, however, the apex partakes in the enlargement, as I have seen it from two to four lines thick, which is double or quadruple the natural size The columnae carneae of the ventricle and of the valves acquire a proportionate enlargement The septum between the two ventricles becomes also notably thickened in the disease of the left ventricle, (which fact seems to mark it as belonging to this rather than the other ventricle,) but never so much so as the other parts

The muscular substance in these cases is of a degree of consistence sometimes double the natural, and is of redder colour The cavity of the ventricle *appears to have lost in capacity what its parietes have gained to* thickness Sometimes I have found this so small, in hearts twice the size of the fist of the individual, as scarcely to be capable of containing an almond in its shell The right ventricle, in such cases, is flattened along the septum, and does not extend to the apex of the heart In extreme cases, it seems as if it were merely included within the parietes of the left ventricle

In hypertrophia of the right ventricle the appearances are somewhat different The thickening is here more uniform, and never so great as in the other I have never found it greater than four or five lines It is always a little greater in the vicinity of the tricuspid valves, and at the origin of the pulmonary artery The columnae carneae are much enlarged, considerably more so, in proportion, than those in the left, in disease of that side Simple enlargement of the right ventricle, without dilatation, is much rarer than that of the left When this disease affects both ventricles at the same time, the only difference from the description just given is, that each side assists to form the apex of the heart

HYPERTROPHIA OF THE LEFT VENTRICLE

It is to this variety of the disease, especially, that the symptoms attributed by M Corvisart to *active aneurism* of the heart, must be referred

These are,—a strong full pulse, strong and obvious pulsation of the heart, absence or diminution of the sound afforded by percussion on the region of the heart, and a tint of complexion rather red than violet. None of these symptoms, however, are constant, and it is not uncommon to find the disease in persons who have none of them. The pulse, in particular, is very deceptive, being almost as frequently weak as strong, in such cases.

The cylinder furnishes signs which are much more constant and positive. The contraction of the left ventricle, examined between the cartilages of the fifth and sixth ribs, gives a very strong impulse, and is accompanied by a duller sound than natural, it is more prolonged in portion as the thickening is more considerable. The contraction of the auricle is very short, productive of little sound, and, consequently scarcely perceptible in extreme cases. The pulsation of the heart is confined to a small extent, being, in general, scarcely perceptible under the left clavicle, or at the top of the sternum, sometimes it is confined to the point between the cartilages of the fifth and seventh ribs. In this disease the patient experiences, more constantly than in any other, the sensation of the action of the heart, but he is less subject to violent attacks of palpitation, except from accidental causes, such as moral affections and violent bodily exertion. In this case, during the palpitations, irregularity and intermission of the pulse are uncommon. There is rather increase of the power of the ventricles than of the noise produced by their action.

HYPERTROPHIA OF THE RIGHT VENTRICLE

According to M Corvisart the symptoms are the same as when the disease is on the other side, only that respiration is more oppressed, and the colour of the face is deeper. Lancisi has mentioned the swelling of the external jugular veins, with a pulsation analogous to that of an artery, as a sign of the aneurism of the right ventricle. M Corvisart has rejected this symptom, because, he says, "it has been found in cases where the left side of the heart was dilated, and because the pulsation may be confounded with that of the carotids." In this opinion I differ from M Corvisart. I have uniformly found this symptom in every case of this kind, of any degree of severity, and I have never met with it in hypertrophia of the left ventricle unless there existed, at the same time, a similar affection of the right. I think a very little attention must distinguish this pulsation from that of the carotids. I would, therefore, be disposed to regard this symptom as one which ought to lead us to suspect the existence of the thickening of the right ventricle.

The contractions of the heart, as explored by the cylinder, give the same results nearly, whether the hypertrophia be on the right or left side, only, in the former case, the shock of the heart's action is greater at the

bottom of the sternum than between the cartilages of the ribs, which is the reverse of what happens when the disease is in the left side of the organ In most men, in health, the heart is heard equally in both these places, and I am disposed to believe, when heard better below the sternum, we may suspect an incipient hypertrophia or dilatation of the right ventricle When both ventricles are affected, the symptoms of both co-exist, only those of the right side are almost always more marked

OF DILATATION OF THE VENTRICLES*

This disease of the heart, which has been named *passive aneurism* by M Corvisart, consists in dilatation of the cavities of the ventricles, with decreased thickness of their parietes With these conditions there are commonly conjoined a notable degree of softening of the muscular substance, and a colour, either more violet, or paler, than natural Sometimes the softness is so considerable, especially in the left ventricle, that the muscular substance can be destroyed by mere pressure between the fingers, and the parietes of the same ventricle may be so much diminished in thickness, as to be only two lines in the thickest point, and scarcely half a line at the apex, while the right ventricle is sometimes so completely extenuated, as to appear merely composed of a little fat and its investing membrane The columnae carneae, particularly of the left ventricle, are more remote than in the natural condition of the part The septum between the ventricles loses less of its thickness and of its consistence than the rest of the parietes

Dilatation may be confined to one ventricle, although it more commonly affects both at the same time When one only is affected, the apex of it extends below the other, but not in so remarkable a degree as in the case of hypertrophia The augmentation of the cavity seems to be more in its breadth than length This is particularly observable when both the ventricles are dilated at the same time, as in this case, the heart assumes a rounded shape, being nearly as wide at the apex as at the base

DILATATION OF THE LEFT VENTRICLE

The symptoms of this affection, according to M Corvisart, are—"a soft and weak pulse, and feeble palpitations —the hand applied to the region of the heart feels as if a soft body elevated the ribs, and did not strike these with a sharp and distinct stroke "

The only certain sign of the existence of this disease is that given by the stethoscope, viz the clear and sonorous contractions of the heart between the cartilages of the fifth and seventh ribs The degree of dis-

*Book Third, Chapter 1, Section Second See footnote on page 332 —F A W, 1940

tinctness of the sound, and its extent over the chest, are the measure of the dilatation thus,—when the sound of the contraction of the ventricle is as clear as that of the contraction of the auricle, and if it is, at the same time, perceptible on the right side of the back, the dilatation is extreme

DILATATION OF THE RIGHT VENTRICLE

According to M Corvisart, the state of the pulse and the pulsation of the heart are very nearly the same as in dilatation of the left ventricle, only that the action of the heart is heard somewhat better towards the bottom of the sternum than in the region of the heart More certain symptoms he considers to be—a greater degree of oppression, more marked serous diathesis, more frequent haemoptysis, and a more livid state of the countenance,—than in the affection of the left ventricle With regard to the swollen state of the jugulars without pulsation, which M Corvisart considers of little importance, I am disposed to look upon it as the most constant and characteristic of the *equivocal* signs of this affection The only constant and truly pathognomonic symptom, however, is the loud sound of the heart perceived at the bottom of the sternum, and between the cartilages of the fifth and seventh ribs of the right side The degree of dilatation is measured by the extent of the action of the heart over the chest The palpitations which accompany this affection consist principally, in an increase of the frequency and sound of the contractions, while, at the same time, the impulse of the heart's action is frequently feebler than in the ordinary state of the patient

OF DILATATION COMBINED WITH HYPERTROPHIA OF THE VENTRICLES*

This reunion, which constitutes the *active aneurism* of M Corvisart, is extremely common, much more common than simple dilatation, and still more so than simple thickening without dilatation This complication may exist in one or both ventricles In the latter case the heart acquires a prodigious size, sometimes more than triple that of the hand of the individual As the augmentation of volume is here the effect of dilatation and thickening, the muscular substance acquires the great firmness already described The apex of the heart becomes blunter, but this is rarely so great as to give to the organ the rounded form noticed in the case of simple dilatation

Dilatation of one ventricle is sometimes conjoined with hypertrophia of the other, but this is not so common as the complication in individual ventricles I have met with the following varieties of this complication 1st, Hypertrophia with the dilatation of the left ventricle, and simple dilatation of the right, 2nd, Hypertrophia with dilatation of the left ventricle,

*Book Third Chapter 1, Section Third See footnote on page 332 —F A W 1940

and simple hypertrophia of the right, 3rd, Hypertrophia with dilatation of the right, and simple dilatation of the left, 4th, Hypertrophia of the right, with dilatation of the left this last is the rarest I do not remember to have met with hypertrophia of the left ventricle (with or without dilatation) complicated with dilatation of the right I would even be disposed to consider such a union as impossible

In this case there is a combination of the symptoms of two affections The contractions of the ventricles yield at the same time a strong impulse and a very marked sound, and they are felt widely over the chest When palpitation is present, the hand applied to the region of the heart is forcibly raised Even in the absence of palpitation, if we observe the patient, we find his head, limbs, and even his bed-clothes shaken at each contraction of the heart The beating of the arteries is often visible

DILATATION OF ONE OF THE VENTRICLES WITH HYPERTROPHIA OF THE OTHER

The signs of this complication are—a mixture of those common to each affection, with predominance of those belonging to the one of greater intensity They are to be discovered by comparing the two sides of the heart together In this case, however, the indications of the cylinder must be taken in conjunction with those of the general symptoms of disease, else we shall be led into error

*DILATATION OF THE AURICLES**

Dilatation of the auricles is an extremely rare disease, and it appears still more so compared with the frequency of the same affection of the ventricles Sometimes we find in subjects affected with hypertrophia or dilatation of the ventricles, the auricles also proportionably enlarged, it is, however, much more common to find these retaining their natural size even in cases where the ventricles are enormously enlarged Sometimes also, but more rarely still, the auricles are dilated when the ventricles are of the natural size

Before we can judge of the extent of this affection we must have precise ideas respecting the natural proportion of the various cavities of the heart As far as the *cavities* are concerned, we must admit that they are very nearly of equal size, but as the parietes of the auricles are much thinner than those of the ventricles, the former, when simply full and not distended, compose only about one-third of the whole organ,—in other words, the size of the auricles is about one-half that of the ventricles Both the auricles have the same capacity, although some anatomists have considered the right

*Book Third Chapter 1 Section Fourth See footnote on page 332 —F A W, 1940

larger, no doubt misled by the greater length of its sinus, and more especially by the distended condition in which it is commonly found after death. A similar distention, though more rarely, takes place also in the left auricle, and this accidental and temporary enlargement is sometimes so considerable, owing to the great extensibility of the auricular structure, as almost to equal the size of the ventricles. In order to distinguish the real from the factitious dilatation, we have only to empty the auricles through the vessels that enter into them, when, in the latter case, these cavities will immediately resume their natural size, and, in the former, they will still nearly retain their acquired volume. There is likewise another mark by which we can at once discriminate the enlargement produced by the accumulation of blood during the few last hours of life, from the permanent increase of capacity of the auricles. In the first case, the parietes of the auricles are greatly distended by the contained blood, and the colour of this appears through the thinnest portions, while, in the latter, the auricles although very voluminous, are still capable of containing more blood, and their parietes remain opaque.

I have never met with decided dilatation of the auricles without some thickening of their walls, and, on the other hand, I have never seen thickening of their walls without an augmentation of their capacity. I may here remark that it requires much experience to judge correctly of hypertrophia of the auricles, as, owing to their great natural thinness, a considerable increase (say double the natural thickness, and the increase is rarely so much) is not obvious to a person little accustomed to such examinations.

The most common cause of dilatation of the left auricle is the contraction of the orifice between it and the ventricle, in consequence of cartilaginous or bony induration of the mitral valve, or of caruncles on its surface. The same causes sometimes occasion the retraction of this valve, and consequently, the permanent patency of the auriculoventricular orifice. In this case dilatation and thickening may arise from the mere action of the ventricle on the auricle. I have never seen any change in the auricles without some alteration in the valves. Dilatation of the right auricle is most commonly the consequence of thickening of the right ventricle. The diseases of the lungs which M. Corvisart reckons among the ordinary causes of this dilatation, seem to me to produce, in general, merely the accidental distention above noticed.

The symptoms of this affection are obscure. M. Corvisart does not distinguish them from those of the corresponding ventricle. I have not myself had yet sufficient experience of the use of the stethoscope in this affection, to speak confidently on the subject. I think, however, there can be little doubt that the signs afforded by it must be confounded with those arising from the disease of the ventricles, or of the valves, of which the auricular affection is the consequence.

Of partial dilatation of the heart, and of the induration of its substance, I have nothing to say in this place

PARTIAL DILATATION OF THE HEART[*]

M Corvisart found, in the person of a young negro who died from suffocation, a partial dilatation of the left ventricle which was truly aneurismatical "On the superior and lateral part of this ventricle there was a tumour almost as large as the heart itself—The interior of this tumour contained several layers of coagulated blood, very dense, and exactly like those found in aneurisms of the limbs The cavity of this tumour, communicated with the ventricle by a small opening, smooth and polished "[†] A similar case is cited by M Corvisart from the *Miscell Nat Curios* I have myself never met with anything of the kind

There is another rare species of dilatation described by Morand,[‡] a second case of which was communicated by me to the Soc de la Facult de Med [§] This is a dilatation formed in the middle of one of the lips of the mitral valve, resembling a thimble, or glove-finger projecting into the auricle

There is still one other variety of partial dilatation of the heart, which I have several times met with, and which is probably, in a great measure, the result of original *malformation* In the natural conformation of the heart, the right ventricle seems to consist of two distinct parts united together, the one of which descends towards the apex of the heart, while the other, almost at right angles to the former, is directed to the left side, and forwards towards the pulmonary artery The dilatation to which I now allude, seemed to exist in both these divisions, while the point of union of the two retained its natural dimensions It is, however, more common to find the anterior or pulmonary division of the ventricle dilated without the other portion, and in every case of dilatation of this ventricle, the former portion is always more dilated than the other This difference becomes still more evident when the dilatation is conjoined with a certain degree of thickening, as, in this case, the pulmonary portion of the ventricle frequently acquires such a degree of firmness that its parietes do not collapse when laid open, a thing which hardly ever happens to the lower portion of the ventricle

INDURATION OF THE HEART[‖]

I have already observed that, in thickening of the heart, the muscular substance possesses an unusual degree of firmness and consistence Corvisart has seen this so great, that the heart sounded like horn when struck,

*Book Third Chapter 1 Section Fifth See footnote on page 332—F A W, 1940

†Op Cit p 283

‡Hist de l acad des Sc 1729

§Bulletin, No 14

‖Book Third Chapter 1, Section Sixth See footnote on page 332—F A W, 1940

and the scalpel experienced great resistance in cutting it However, the muscular substance of the heart "retained its natural colour, and did not appear to be converted either into the bony or cartilaginous tissue" I have never met with this species of induration, although M Corvisart has several times I consider it as the last degree of hypertrophia

SOFTENING OF THE HEART*

I have already noticed this condition of the heart In it the muscular substance is sometimes so soft as to be almost friable, the fingers passing easily through the parietes of the ventricles Whatever may have been the patient's disease, the heart is rarely filled with blood, and the ventricles equally collapse whatsoever may be their varying thickness This affection of the heart is almost always attended by some change of colour in the organ Sometimes this is deeper, and even quite violet, and this is particularly the case in fevers of the kind named *adynamique* by Pinel More commonly, however, the softening of the heart is attended by a striking loss of colour, so as to resemble the palest dead leaf This pale or yellowish tint does not always occupy the whole thickness of the heart, sometimes it is strongly marked in the central portions, and very little on the exterior or interior surfaces Frequently the left ventricle and interventricular septum exhibit this appearance, while the right ventricle retains its natural colour, and even a degree of firmness greater than natural Again, we sometimes find here and there spots of the natural colour and consistence in hearts which are, everywhere else, much softened and quite yellowish This variety of yellowish softening is particularly observable in those cases where dilatation is conjoined with a slight degree of thickening It is also found in simple dilatation, although it is more common to find this state accompanied by that species of softening which is marked by an augmentation of the natural colour of the organ There is a third variety of softening of the heart, which will be noticed in another place, and which is attended by a pale white colour of the muscular substance In this, the degree of softening never reaches that of friableness, often it is scarcely perceptible, but the parts are flabby, and the parietes of the ventricles quite fall together on being opened This condition will be noticed under the head of inflammation of the pericardium, as it is peculiar to that disease

It would seem that the softening of the heart discovered in subjects whose death has been very gradual, is an acute affection, it is evidently still more so where it exists only partially in the substance of the organ On the contrare, in cases where the heart is softened and yellowish throughout, it is probable that the affection has existed for a long time The deep-coloured softness observed in subjects dead of fever, may, I think be compared to that adhesive softness of the other muscles often observed in these cases, and which is also accompanied by a degree of redness greater than

*Book Third Chapter 1 Section Seventh See footnote on page 332 —F A. W, 1940

natural. This softening of the heart, as well as the analogous *gluey* or *fishy* (gluant ou poisseux) state of the muscles, is particularly observable in putrid fevers, particularly when these exhibit the phenomena formerly considered as marks of putridity—viz. livid intumescence of the face, softening of the lips, gums, and internal membrane of the mouth, black coating on the tongue and gums, earthy aspect of the skin, distended abdomen and very fetid dejections. I cannot assert that this softening of the heart exists in all kinds of continued fevers, but I have met with it constantly in such cases as I have attended to. Could it account for that frequency of pulse which exists, sometimes for several weeks in convalescence from fevers, although the patient continues to regain flesh and vigour?

Cases of total softening of the heart are usually accompanied by a certain degree of cachexy, even when the individuals are otherwise in tolerable health. When such subjects are attacked with dilatation or hypertrophia of the heart, as almost always happens, they do not present the usual swollen and livid state of the face observable in other cases of this sort.

When softening exists along with dilatation of the ventricles, the sound produced by the contraction of these cavities, although loud, is yet dull, and without the clearness which attends common dilatation. When it is complicated with hypertrophia, the sound of the contraction of the ventricles is so obtuse as to be nearly inaudible, and in extreme cases, the impulse of the heart is attended by no noise whatever.

ATROPHY OF THE HEART

It is an important question whether the heart be susceptible of diminution of size and power like other muscles, and, if so, whether this affords any hope of cure, by debilitating measures, in cases of hypertrophia. This much is certain, that, in cases where there is much emaciation, as in Phthisis and Cancer, the heart is generally found small. From this consideration, I have in many cases of hypertrophia attempted the method of cure proposed by Valsalva in aneurism. Almost all my patients got shortly tired of the extreme severity of the regimen, and alarmed by the frequency of the bleedings. In three cases, however, I have been so far successful that I am led to believe that this disease is not entirely beyond the resources of art and nature. Two of these were young women, the one twelve and the other eighteen years of age, both of whom presented symptoms of hypertrophia in a high degree. The privation of one-half of their ordinary diet, and some occasional general and local bleedings effected the gradual diminution, and, eventually, the complete cessation of all their symptoms. The youngest has now been cured for four years, and has long ago returned to her usual regimen. The other still follows the prescribed regimen, and is

*Book Third. Chapter 1, Section Eighth. See footnote on page 332.—F. A. W., 1940.

now quite reconciled to the diminished quantity of food. Blood-letting has not been found necessary for the last year, and the general symptoms of the disease have disappeared, although the unnatural thickness of the parietes of the heart is still recognizable by stethoscope. The third case is still more conclusive, as I have been enabled to ascertain the state of the heart after death. I shall therefore state it more particularly.

Case 42. A woman, fifty years of age, had been affected for twelve years with all the symptoms of disease of the heart, in a very high degree, viz. strong and frequent palpitations, habitual dyspnoea, breathlessness on using the least exercise, sudden startings from sleep, almost constant edema of the lower extremities, and lividity of the cheeks, nose, and lips. These symptoms had increased during the last year, so that she could scarcely move from her chair without the feeling of suffocation. In this state I recommended the treatment of Valsalva, which she agreed to. I immediately reduced her aliments to one-fourth of her former allowance, and bled her once a fortnight, either from the arm or by leeches. This mode of treatment gave immediate relief, and in the course of six months all the symptoms had disappeared, and, with the exception of debility (which however was not greater than it had been previously), she enjoyed a better state of health than for many years before. The respiration was now free, and the palpitations, oedema, startings, and lividity of the face had quite disappeared. After this I recommended the bleedings to be decreased in frequency, and I dispensed with them altogether at the end of a year. She also returned gradually to her old regimen, only that now a much smaller quantity of food satisfied her appetite. She lived two years in a state of perfect health, when she was suddenly carried off by an epidemic cholera. Upon examining the body after death I found the heart considerably less than the closed hand of the individual, being only about the usual size of that of a child twelve years old, although this woman was five feet three inches in height. The exterior of the heart resembled, in appearance, a withered apple, the wrinkles running longitudinally. The ventricular parietes were flaccid, but without any notable softening, and of the natural thickness. I am well aware that nothing can be deduced from a single case, but I have thought the above relation might be useful by stimulating others to prosecute this subject more at length.

FATTY DEGENERATION OF THE HEART*

In medical writings we find many examples of the heart being overloaded with fat in a surprising manner, and to which change of structure various symptoms, and even sudden death of the individuals, were attributed. M. Corvisart thinks that an enormous accumulation of fat around the heart may, in fact, produce such effects, although he has met with no similar, or other permanent derangement, in persons whose hearts were

*Book Third, Chapter 1. Section Ninth. See footnote on page 332.—F. A. W. 1940.

found to be much loaded in this manner I have also met with a great many cases of hearts, overloaded in this manner, in subjects dead of various diseases In these the fat was deposited between the muscular substance of the heart and the investing pericardium, and chiefly at the union of the auricles and ventricles, at the origin of the great vessels, and along the tract of the coronary arteries, also along the two edges and at the apex of the heart Sometimes the posterior face of the right ventricle is covered by this deposition in its whole extent, a circumstance which rarely has place on the surface of the left ventricle

The fatter the heart is, the thinner, in general, are its parietes Sometimes these are extremely thin, especially at the apex of the ventricles and the posterior side of the right ventricle On examining ventricles affected in this manner, they present the usual appearance internally, but on cutting into them from without, the scalpel seems to reach the cavity without encountering almost any muscular substance, the columnae carneae appearing merely as if bound together by the internal lining membrane In these cases the fat does not appear to be the product of degeneration of the muscular fibres, as these can be separated by dissection Sometimes, indeed, portions of fat penetrate deeply between the muscular fibres, but, even in this case, the distinction between the two tissues is still very marked, and they are confounded by no mutual gradation of colour or consistence It would seem probable from this, that, from pressure or some unknown aberration of the powers of nutrition, the muscular substance has wasted in proportion as the investing fat has increased It would seem reasonable to expect rupture of the heart from an affection of this kind, such an instance, however, has never occurred to me Very commonly we find, in such subjects, a large quantity of fat in the lower part of the mediastinum, particularly between the pericardium and pleura This fat, much reddened by its small vessels, and covered by its pleura, assumes the figure of a cock's comb and is firm The fat surrounding the heart, on the contrary, is almost always of a pale yellow colour I have not observed, any more than M Corvisart, any symptoms that could directly denote the existence of an accumulation of this sort I apprehend it must exist in a very great degree before it gave rise to any serious complaint This is not, therefore, the condition I wish to denote by the name of *Fatty degeneration of the Heart* This latter is an actual transformation of the muscular substance into a substance possessing most of the chemical and physical properties of fat It is precisely similar to the fatty degeneration of the muscles observed by Haller,* and Vicq-d'Azyr † I have only met with it in a small portion of the heart at one time, and only towards the apex In these portions the natural red colour is superseded by a pale yellow like that of a dead leaf This change of structure appears to proceed from without inwards Near the internal surface of

*Opusc Pathol
†Tom V

the ventricles, the muscular texture is still very distinguishable; more externally, it is less so; and still nearer the surface it becomes gradually confounded, both in colour and consistence, with the natural fat of the apex of the heart. In such cases, however, even the portions that still retain most of the muscular character, when compressed between two pieces of paper, still grease these very much. This character distinguishes this species of degeneration from simple softening of the viscus. I have never found rupture of the heart attributable to this change, any more than to the morbid accumulation of fat. It is denoted by no symptoms with which I am acquainted.

CARTILAGINOUS OR BONY INDURATION OF THE MUSCULAR SUBSTANCE OF THE HEART*

I have never met with ossification of the muscular substance of the heart, and only a small number of examples of this are on record. M. Corvisart found, in the case of a man who died of hypertrophia of the left ventricle, the whole apex of the heart, and more partially the columnae carneae, converted into cartilage. (Op. cit.)

Haller (Opusc. Pathol.) found, in a child, whose heart was of the natural size, the inferior part of the right ventricle, the most muscular parts of the left auricle, and the sigmoid valves of the aorta and pulmonary artery, in a state of ossification. M. Renauldin has published, in the Journal de Med. for 1816, a very interesting case of the same kind. The patient was a man thirty-three years of age, much addicted to study, and subject to violent palpitations on the slightest motion. "On applying the hand to the region of the heart a sort of motion of the ribs was felt, and even the slightest pressure produced very acute pain, which lasted long after the pressure was discontinued. On examining the body after death the heart was found extremely hard and heavy. On attempting to cut the left ventricle great resistance was found, owing to the total conversion of the muscular fibre into a sort of *petrifaction*, having in some places a sandy character, in others a resemblance to saline crystallization. The grains of this species of sand were very contiguous to each other, and became larger towards the interior of the ventricle. They were continuous with the columnae carneae, which were themselves converted into a similar substance, but still retained their original form, only much enlarged. Some of these sabulous concretions were of the size of the point of the little finger, and resembled small stalactites shooting in different directions. The ventricle was thickened. The right ventricle and great arterial trunks were sound. The temporal and maxillary arteries, and also a part of both the radial arteries were ossified." We frequently find on the interior surface of the ventricles, especially the left, cartilaginous scales continuous with

*Book Third. Chapter 1. Section Tenth. See footnote on page 332.—F. A. W., 1940.

the lining membrane, and apparently deposited between it and the muscular substance of the heart These are generally small I have never found them ossified

*OF CARDITIS**

Inflammation of the heart is a rare affection, and is, consequently very imperfectly known both in a practical and pathological view There are two varieties of it, the general, or that affecting the whole heart, and the partial, or that confined to a small extent of it There perhaps does not exist on record a satisfactory case of general inflammation of the heart, either acute or chronic The greater number of cases so called, and particularly those given by M Corvisart, are evidently instances of Pericarditis attended by that degree of discoloration of the heart which we shall find frequently to accompany that affection Nothing proves that the paleness of *the heart in such cases is the consequence of inflammation The affection* generally increases both the redness and density of the parts which it occupies,—but the discoloration in the cases alluded to is conjoined, in general, with a perceptible softening of the heart It is further observable that, in these cases, pericardium was filled with pus while not a particle was found in the substance of the heart itself, now, pus must be considered as the most unequivocal indication of inflammation The only case which I have met with of general inflammation of the heart possessing this unequivocal mark, is noticed by Meckel in the Mem de l'Acad de Berlin But this case is described with so little precision, as merely to prove the possibility of the fact, and affords no help towards a general description of the disease

Instances of partial inflammation of the heart, characterized by the presence of an abscess or ulcer in its parietes, are much more common Bonetus has recorded a good many such cases in his *Sepulchretum* I have only met with one instance of the kind In this (in a child twelve years old) the abscess was situated in the parietes of the left ventricle, and might have contained a filbert it was complicated with pericarditis In another case, of a man of sixty years old, I found an albuminous exudation, of the consistence of boiled white of egg, and of the colour of pus, deposited among the muscular fibres of the left ventricle The patient had presented symptoms of an acute inflammation of some of the thoracic viscera, without precisely indicating its site Orthopnoea, and a feeling of inexpressible anguish, had been the chief symptoms

Ulcers of the heart have been still more frequently observed than abscess, they have been met with in its external and internal surface † All the cases, however, recorded under this name are not quite correctly designated In the *Sepulchretum* we frequently find a case of pericarditis, attended with a rough and uneven pseudo-membranous exudation, mistaken

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*Book Third Chapter 1 Section Eleventh See footnote on page 332 —F A W 1940
†Morgagni, Epist XXV

for an ulcer of the exterior surface of the heart This has been noticed by Morgagni (Epist 20 and 25) That true ulcers of this surface, however, have been observed, is beyond doubt A case of this kind is described by Olaus-Borrichius in the following words "Cordis exterior caro, profunde exesa, in lacinias et villos carneos putrescentes abierat,"* and similar cases are recorded by Peyer† and Graetz‡ Ulcers on the interior surfaces of the heart are perhaps more common than on the external, or, at least, there are on record a greater number of incontestible examples of the former Bonetus, Morgagni and Senac have collected a great many of these I have myself only met with one case of this kind The ulcer was on the internal surface of the left ventricle, and was an inch long by half an inch wide, and was more than four lines deep in its centre This patient had laboured under hypertrophia of the left ventricle, which had been recognised before death this was occasioned by rupture of the ventricle This terrible and, fortunately, very rare accident, is almost always the result of ulceration of the ventricular parietes Morand has collected several cases of this kind in the Mem de l'Acad des Sciences for the 1732, and Morgagni has described a similar instance—(Epist 27)

Rupture of the heart from violent exertion, without previous ulceration, is much rarer still, and the number of incontestible examples of this is very small Several cases, recorded as such, are so imperfectly described, as to leave a doubt whether the alleged rupture might not have been rather the consequence of the incisions of an inexpert dissector The best authenticated examples of this kind of rupture are those given by Haller (Elem Physiol) and Morgagni (Epist 27)

It is surprising that the great thinness of the parietes of the ventricles, in the cases of accumulation of fat, does not give rise to rupture, more especially towards the apex and posterior part of the right ventricle This is, however, so far from being the case, that ruptures of the right ventricle are much rarer than those of the left, and that, in this last, the rupture, when it occurs, is very rarely towards the apex

M Corvisart has given, for the first time, examples of another species of rupture of the heart, of a less certainly dangerous nature,—that, namely, of the tendons and fleshy pillars of the valves §

In the three cases related by him the rupture appears to have been the consequence of violent efforts in lifting great weights, etc A sudden and very intense feeling of suffocation was the immediate result of this accident, which terminated in exhibiting all the usual symptoms of disease of the heart I shall have occasion to notice in a subsequent section a case of the same kind, only produced, apparently, by ulceration of the tendons

*Sepulchret Lib II Obs 86
†Ibid Sect II Obs 21
‡Disput de Hydr pericard Sect 2
§Corvisart on the Heart Obs 33, 40 and 41

In the present state of our knowledge it is impossible to ascertain the existence of either an abscess or ulcer of the heart

*OF CARTILAGINOUS AND BONY INDURATION OF THE VALVES OF THE HEART**

The mitral and sigmoid valves of the aorta are subject to become the site of cartilaginous or bony productions, which increase their thickness, alter their shape, and obstruct, sometimes almost totally, the orifices in which they are placed The tricuspid and sigmoid valves of the pulmonary artery are much less subject to these alterations, although they are not quite exempt from them, as Bichat thought Morgagni found (Epist 37), in the case of an old woman, both these partially indurated He likewise found, in a young woman, the sigmoid valves of the pulmonary artery agglutinated by means of a cartilaginous induration, partly ossified, so as considerably to diminish the diameter of the artery M Corvisart has twice met with a cartilaginous induration of the base of the tricuspid valve, and I have myself sometimes observed slight cartilaginous incrustations, both at the base, and on the points of this valve I am not, however, aware that any one has found these indurated portions completely ossified, nor do I believe that the induration has ever been so considerable as to occasion a serious state of disease For these reasons I shall confine my remarks to the valves of the left ventricle

The cartilaginous induration of the mitral valve is sometimes confined to the fibrous bands found in its base In this case it has the appearance of a very smooth, though unequal roll, lessening the orifice in which it is situated This sometimes has the consistence of perfect cartilage, sometimes only that of imperfect cartilage Similar incrustations sometimes are met with in other parts of these valves The bony indurations present the same characters as to situation and inequality of thickness Though formed in the interior of the valve, they often project from it quite uncovered These ossifications are never perfect bone, they are whiter and more opaque, more fragile, evidently contain a greater proportion of phosphat of lime On this account they have been frequently named *stones* or *calculi* In fact, they frequently bear a striking resemblance to small pieces of stone, of very irregular surface, recently broken When they are situated in the floating extremities of the valve, these are sometimes united together, so as to reduce the orifice to a mere slit, which will, sometimes scarcely admit the blade of a knife or a goose-quill M Corvisart found the orifice between the auricle and ventricle reduced to a channel three lines wide, and bent like the canalis caroticus, from the thickening of the ossified mitral valves Sometimes, though rarely, the tendinous cords of the mitral valve are affected in the same manner, and M Corvisart in one case found the whole of one of its pillars ossified †

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*Book Third, Chapter 1 Section Twelfth See footnote on page 332 —F A W, 1940

†Op cit, p 212, 214

The ossification of the sigmoid valves of the aorta may commence, like that of the mitral, in their base of their loose edges,—and much more frequently in one of these situations than in the intermediate portion When in the loose extremity, the ossification seems most frequently to originate in the small tubercles known by the name of the *Corpora Sesamoidea*

When the ossification is confined to the floating edge of the valves, or when the base though ossified is little thickened, the valve may still perform its functions, provided the middle portion of it be still sound But when the ossification is extensive, the valves grow together, and get incurvated, either towards their concave or convex side, so as to acquire the appearance of certain shells In this state they are immoveable, being either fixed on the side of the aorta, or in the orifice of the ventricle Very frequently, of the three valves one is bent in a direction opposite that of the two others In one case, M Corvisart found all the three valves ossified in their closed position so as to leave merely an extremely small slit for the passage of the blood The evil of this was partly obviated by one of the valves, although ossified and very thick, still retaining, at its base, sufficient mobility to allow an increase of one or two lines to the orifice during the action of the heart

The symptoms of ossification of the mitral valve are somewhat different from those attending the same affection of the sigmoid According to M Corvisart the principal sign of the former lesion is "a peculiar rustling sensation, perceived on the application of the hand to the region of the heart " I have often noticed this symptom, which is very readily recognized after being once perceived, although it is difficult to give a description of it The nearest idea I can give of it is by comparing it to the purring of a cat when pleased The same sort of quality is said, by M Corvisart, to exist in the pulse, which, he adds, is weak, but without hardness or fullness To these symptoms may be added those characteristics of hypertrophia and dilatation of the left auricle and whole right side of the heart, which usually follow the affection of the valve

I must confess that I have never perceived the peculiar character of the pulse described by M Corvisart, and that I have frequently found wanting the peculiar vibration in the region of the heart in cases of undoubted disease of the valves I believe the latter sensation is only perceptible by the hand when the contraction of the orifice is very considerable In ossification of the sigmoid valves, several signs deduced from the state of the circulation are given by M Corvisart, but the whole may be reduced to the *purring* sensation above mentioned

Since I have used the cylinder I have only met with three cases of ossification of the mitral valve accompanied by the purring sensation, and only four cases of the same affection of the sigmoid in a slight degree, and unattended by the purring In comparing these, however, with the nu-

merous cases I had before studied, I think I can give the following results, if not correct, as, at least, approaching to correctness

Ossification of the mitral and sigmoid valves does not produce irregularity of the circulation, and cannot therefore be suspected from the state of the pulse, or by the application of the hand to the region of the heart, unless it is so considerable as materially to lessen the orifices of the left ventricle In ossification of the mitral valve, in a middling degree, the sound which attends the contraction of the auricle becomes much more prolonged, more dull, and with something in its tone which reminds one of the rasping of a file on wood, and sometimes of a bellows smartly compressed This sound is well-marked when the *purring* is not perceptible to the hand, but it is much more distinct when this is perceptible, and is, indeed, proportional to its intensity

The ossification of the sigmoid valves of the aorta is shewn by the existence of this sound during the contraction of the ventricle, but this does not exist in slight degrees of the affection, nor in a similar condition of the mitral

In these cases, as in dilatation and hypertrophia, the alternate examination of the heart under the sternum and between the cartilages of the fifth and seventh ribs, as well as the state of the external jugulars, will always enable us to decide in which side of the heart the disease exists

*OF ACCIDENTAL OR EXTRANEOUS PRODUCTIONS IN THE HEART**

Of all the organs of the body the heart is perhaps the least liable to these productions, if we except ossifications Twice only have I found tubercles in its muscular substance, and not once melanosis, medullary sarcoma, or any other species of cancer M Recamier, however, informs me that he has found the heart partially converted into a scirrhous matter resembling lard, in a person who also had cancers in the lungs In the *Sepulchretum* we find several examples of tumours in the heart, which appear to have been cancerous Columbus found two hard tumours of the size of an egg in the parietes of the left ventricle † Morgagni relates a case where there were numerous small tubercles on the external surface of the right auricle, in a subject which exhibited similar, but larger, tumours in the mediastinum, lungs, lymphatic glands, and cellular substance of the thorax and abdomen (Epist 78)

Encysted serous tumours are equally rare in this viscus When they do occur they are most commonly found between the muscular substance and investing pericardium Examples have been recorded by Baillou, Houlier, Cordaeus, Rolfinckius, Thebesius, Fanton, Dalsalva, Morgagni, and Dupuytren The latter found cysts of this kind in the parietes of the right auricle, projecting inwardly, and distending it to a size equal to the whole of the

*Book Third Chapter 1, Section Thirteenth See footnote on page 332 —F A W, 1940
†De re Anat lib XV

other parts of the heart Morgagni describes a tumour, which was evidently a hydatid, implanted on the surface of the left ventricle (Epist 21), and which appears to have been that variety named by Rudolphi *Cysticercous finnus*

OF POLYPI OF THE HEART*

It was formerly customary to attribute to the polypous concretions of the heart observed after death, the symptoms which truly depend on the enlargement of that organ The incorrectness of this opinion is proved by the fact, that these concretions are very frequently found in persons who have never exhibited any symptom of disease of the heart in truth, they are met with in three-fourths of dead bodies It is equally erroneous to believe, with some modern authors, that polypi never begin to form until the moment of death Many facts prove that these concretions can be formed during life The phenomena of aneurisms alone prove this Haller found the carotid artery and internal jugular vein quite obstructed by very firm concrete fibrine in one case, and the inferior vena cava in another † Vinckler,‡ Stancari and Banaroli have met with similar cases §

I have myself observed, in a consumptive subject, an obliteration of the inferior cava for the space of four fingers' breadth This obstruction was produced by a whitish fibrinous concretion which filled the whole caliber of the vein The exterior layers of this concretion were like the buffy coat of the blood, only much firmer, and adhered strongly to the inner coat of the vein, the inner portions were, on the contrary, of a yellowish colour, more completely opaque, and of a friable character like certain kinds of cheese In another case I found a similar obstruction in the carotid artery, and, in a third, I observed the whole of the vessels of the pia mater, in a circumscribed space about the size of the palm of the hand, injected with a similar concretion None of these individuals had exhibited any symptoms indicative of such an affection, nor did there exist in any of them any obstacles to the course of the blood which might account for them we must, therefore, attribute them to spontaneous coagulation of the blood, and reasoning, a priori, therefore, nothing is more probable than that the blood may coagulate during life, in the heart also, at least at the very close of life, when the circulation is performed only in an irregular and imperfect manner M Corvisart was therefore correct in distinguishing polypi into such as are of a formation posterior to death, and such as have been produced while the individual was still alive These two kinds are easily distinguished from each other The former, or those of recent formation, exhibit merely a slight layer of whitish opaque fibrine partially enveloping the coagula of blood contained in the heart and large vessels This fibrinous or buffy layer never completely surrounds the coagula, and does not adhere

*Book Third Chapter 1 Section Fourteenth See footnote on page 332—F A W 1940

†Opusc Pathol obs 23 24

‡Dissert de Vasor lithias

§Morgagni Epist 64

to the parietes of the heart or vessel in which it is contained. Sometimes this layer is thicker, and, in this case, especially if the subject is dropsical, it is semi-transparent and tremulous like jelly.

On the other hand, the polypi of more ancient formation are of a much firmer consistence, and adhere more or less strongly to the parietes of the heart. In the ventricles and auricular sinuses, this adhesion is partly caused, no doubt, by the intertexture of the concretion with the columnae carneae, but, even here, the principal part of the attachment is independent of any mechanical structure of the parts. These concretions are of a more distinctly fibrinous texture than are the recent formations or the buffy coat of the blood, and they are, further, of a pale flesh or slight violet colour, while the more recent are, as already mentioned, of a white or yellowish colour.

These ancient concretions are found most frequently in the sinus of the right auricle, and in the right ventricle. When in the former, they completely obstruct its cavity, but in the ventricle they only double in thickness its parietes (thereby lessening its cavity) and obstruct the descent of the tricuspid valve. In this case, one may remove all the loose coagulated blood without injuring the concretion, it is even possible that this might be mistaken for the natural boundaries of the cavity.

The columnae carneae to which these concretions are attached, are commonly perceptibly flattened, a circumstance which, of itself, would prove their formation to be anterior to death. M. Corvisart was the first, as far as I know, to observe this flattening of the columnae, in the case noticed by him they were quite *effaced*. I have not met with any case so strongly marked as this, but it is by no means rare to find cases wherein the thing is very perceptible.

There is still a third species of concretion, evidently more ancient than those just described,—of a formation, perhaps, several months anterior to the patient's death. These are found adhering to the parietes of the heart, sometimes so firmly as only to be detached by scraping with the scalpel. Their consistence is less than that of those just noticed, being not at all fibrinous, and resembling rather a dry friable paste or a fat and somewhat soft cheese. They have lost the semitransparency of recently concreted fibrine, and resemble in every respect those layers of decomposed fibrine met with in false aneurisms. I have only met with concretions of this kind in the auricles.

*OF EXCRESCENCES ON THE VALVES AND INTERNAL PARIETES OF THE HEART**

There are two very distinct varieties of this affection. The first has been described by M. Corvisart under the name of *Excrescences of the Valves*, the other, which does not appear to have been hitherto described, I shall

*Book Third Chapter 1, Section Fifteenth. See footnote on page 332.—F. A. W., 1940

notice under the name of *globular excrescence* The first kind might very well be named warty excrescence, inasmuch as they are extremely like warts, especially those of venereal origin on the parts of generation Like these, the excrescences in the heart sometimes resemble small cherries, in their form and tuberous surface, at other times they are elongated into the form of a small cylinder or cord, and, occasionally, they are so short and so crowded together, as merely to give to the parts on which they are situated a rough or rugged surface, more frequently, however, they are either isolated or ranged in a single line along the loose, or the attached border of the valves I have never observed any longer than three lines The colour of these excrescences is sometimes whitish like that of the valves, and hardly so opaque, more commonly they are either wholly or in part tinged with a reddish or light violet colour Their texture is fleshy, like venereal warts, only of somewhat less firm consistence They adhere immediately to the subjacent parts, sometimes so strongly as to be only separable by incision, more commonly they are easily removed by scraping The venereal origin of these excrescences, entertained by M Corvisart, appears to me very improbable, when we consider their rarity and the frequency of venereal complaints, and when we meet with them, as we do, in individuals who, in all probability, never had this disease Whatever may be the remote cause of these bodies, the manner of their formation seems to me more explicable In dissecting the more voluminous excrescences, it has always appeared to me that their texture has borne a strong resemblance to that of the more compact polypous concretions Frequently we observe in their centre a violet or sanguineous tint, and sometimes I have even found a very small, but distinct, coagulum of blood From these circumstances I am led to believe, that these excrescences are merely polypi organized by the same process which transforms the false albuminous membranes into true adventitious membranes, or into cellular substance

In like manner as M Corvisart, I have only met with these excrescences in the following situations, viz the mitral, tricuspid, and sigmoid valves, and (much more rarely) the interior of the auricles, especially the left In general they are more common in the left than the right side of the heart I may here remark that the view of the formation of these excrescences, given above, proves that they are not likely to occur but in subjects already affected with some serious disease of the heart or large vessels, a circumstance, as we shall find, in another place, which must render their diagnosis very difficult [In the following notice of a case of this affection, I shall, as in many of the former cases, omit several of the symptoms with the intention of again noticing them in another part of this work]

Case 43 A man, aged thirty-five, at the period of his coming into hospital, had been affected for five months, with great dyspnoea and violent palpitations on making any considerable exertion, startings from sleep, and

occasional spitting of blood. For a few days past he had laboured under a severe diarrhoea. His countenance was tranquil, with some colour, the pulse small, hard, and regular, and the respiration oppressed. The action of the heart was not quite regular, but there was no distention of the jugular veins. This patient died on the third day.

The pericardium contained half a pint of serum. The heart was double the size of the patient's fist. The right ventricle was very large, its parietes being at least four lines thick, and its columnae very large. The tricuspid valves, and the sigmoid of the pulmonary artery, were of a deep violet red colour. The right auricle was sound. The left ventricle was one-third larger than natural, and its walls were six lines thick, and its columnae very thick. One of the tendons affixed to the edge of the mitral valve was ruptured about its middle. This rupture appeared to have been the consequence of progressive wasting of its middle part, and one of the other tendons of the same valve was unequally extenuated, but still whole. The whole floating border of the mitral valve was covered with small excrescences such as I have described, varying in size, form, and consistence. Altogether they gave to the valve a thickened and fringed appearance. The sigmoid valves of the aorta, and the lining membrane of this artery, were extremely red, and exhibited in this respect a striking contrast with the inner membrane of the ventricle. The whole inner surface, and indeed the whole parietes, of the left auricle, were of the same red colour, and, above the opening of the left pulmonary veins, and about two lines from the auriculo-ventricular opening, there was about an inch square coated with a congeries of excrescences similar to those on the mitral valve, and very firmly attached. The muscular substance of the heart was of moderate firmness. The pleura contained about a pint of serum on each side. The lungs were sound.

The *globular excrescences* have a quite different appearance from those just described, resembling little balls or cysts, of a spherical or oval shape, and of a size from that of a pea to a pigeon's egg. The exterior surface of these is equal, smooth, and of a yellowish white colour, and the thickness of their parietes is very uniform, being never more than half a line. The substance composing their parietes is opaque and very similar to that of ancient polypi, its consistence being firmer than boiled white of egg. The inner surface of these parietes (the cyst) is not so smooth as the exterior, and it appears to be composed of a softer substance, which occasionally has the appearance of passing gradually into the matter contained within it. This matter may exist in three different states, all of which may be found in the same subject, but in different cysts. These are, first, a liquid resembling half-coagulated blood, only turbid as if intermixed with some insoluble powder, and sometimes containing a few clots of perfectly coagulated blood, second, a more opaque matter, of a pale violet colour, of a pultaceous consistence, and very like the lees of wine, and third, yellow

ish, opaque fluid, like thick pus or thin paste I have only met with cysts of this kind in the ventricles and auricular sinuses They are found as frequently in the right as left side of the heart, generally near the apex of the ventricles, and always adherent to the walls of the cavity They are attached by means of a pedicle, which is often so slightly connected with the columnae carneae as to be detached from them without being ruptured This pedicle, although forming part of the excrescence, resembles the common polypi more than the other portions, and seems as if it were of more recent formation and less perfectly organized I have never found these bodies more organized than I have described, and I have considered those containing clots of blood as the newest, those containing a fluid like the lees of wine as next in order, and those containing a puriform matter as the most ancient I have met with these excrescences in subjects dead of different diseases, but all of whom had remained in a dying state (agonie) for several days or even weeks

The only case that I have met with in medical writings, which seems to me to agree with the above description, is recorded in the *Miscel Natur Curios* The affection, nevertheless, does not appear to be extremely rare, as I have met with several cases of it

OF THE RED COLOUR OF THE INTERNAL MEMBRANE OF THE HEART AND LARGE VESSELS*

In examining dead bodies we frequently find the inside of the aorta and pulmonary artery uniformly reddened, as if stained by the blood they contained This colouring is of two kinds,—either bordering on scarlet, or violet The scarlet colour has its seat exclusively in the inner membrane, as, when this is removed, the tunic beneath is found of the natural colour This colour is quite uniform, as if painted, without any trace of vascularity, only sometimes more intense in one place than another Sometimes this stain diminishes progressively from the origin of the aorta, but frequently it terminates quite abruptly with irregular edges Sometimes in the middle of a very red portion we find a circumscribed spot retaining the natural white colour, like the whiteness produced by pressure with the finger on an erysipelatous skin The origin and arch of the aorta are the situations most commonly reddened, and, with them, the sigmoid and mitral valves When the pulmonary artery is affected, its valves, as well as the tricuspid, are commonly in the same state The lining membrane of the ventricles and auricles is frequently colourless when the valves are deeply stained, not unfrequently, however, the auricle participates in the affection, but scarcely ever the ventricles This redness is attended by no sensible thickening of the part, and it entirely disappears after a few hours maceration

M Corvisart has slightly noticed this affection, and has avowed his ignorance of its nature and cause Franck, who has observed it through the

*Book Third Chapter 1 Section Sixteenth See footnote on page 332 —F A W 1940

whole tract of the arteries, considered it as the cause of a particular and uniformly fatal fever. My own observations are far from leading to the same result, although I confess myself ignorant of the nature of this affection. The most natural idea respecting it is, that it is the result of inflammation. But mere redness, without thickening of parts, does not sufficiently characterise this state, while the abrupt termination, and exact circumscription presented by the redness in certain cases, seem not easily to accord with the nature of inflammation. On the other hand, it may, indeed, be said, that in the serous and mucous membranes, this sort of redness by stains is more characteristic of inflammation than the mere sanguineous infarction of the capillaries, which might take place either at, or after, death. The following is an example of this affection.

Case 44. A young woman, flesh-coloured and plump, came into hospital complaining only of intense headache, of three days' duration. At the end of two days the disease assumed the appearance of acute hydrocephalus, the pulse being very slow, very regular and of moderate strength. The cerebral symptoms increasing rapidly, this patient died at the end of ten days from the invasion of the disease, after the application of the usual measures, and particularly the employment of a great number of general and local bleedings indicated by the violence of the headache. For two days before death, the pulse became more frequent, but not stronger, nor more irregular. On examining the body, besides the hydrocephalus, there were found tubercles in the lungs, large tuberculous ulcerations in the intestines, extensive emphysema in several portions of the mucous coat of the intestines, unequivocal marks of confirmed lues, and, finally, a very intense redness of all the valves of the heart, the aorta, and particularly of the pulmonary artery.

One of my pupils informs me that he found in an aorta intensely reddened, some small purulent collections, resembling miliary pustules, situated between the internal and middle coats. This, however, must have been the consequence of disease of the middle coat, itself, as we can hardly suppose that inflammation of the internal coat would terminate in suppuration of its adherent surface. I do not, however, mean to deny the possibility of inflammation of blood-vessels. On the contrary, I think it probable that the affection we have been describing is of this nature, and I would be disposed to consider the various concretions of blood already mentioned, for example, those which produce obliteration of veins, and the warty excrescences, as the result of inflammation.

The second species of redness of the large vessels has a quite different appearance, being, in place of a bright red, of a violet hue. It is also usually extended at the same time to the aorta, pulmonary artery, valves, auricles and ventricles. This variety is not so exactly confined to the lining membrane, as we find the muscular substance of the auricles and ventricles, and even the fibrinous coat of the aorta and pulmonary artery,

participating in it, at least partially I have found this variety of colouring in subjects dead of putrid fevers, emphysema of the lungs, and disease of the heart All these individuals had remained long in a moribund condition, with suffocation, and I have thought that the violent tint was deep in proportion to the intensity and duration of the latter symptom From this circumstance I am disposed to consider this condition of the vessels as the effect of deranged circulation and congestion of the blood in the capillaries, being analogous to the livid hue of the cheeks, etc observable in persons dead of disease of the heart It is, in fact, an effect of death, or at most produced in articulo mortis

I would here beg to observe, that it is often difficult to distinguish mere congestion of the capillaries from actual inflammation The distinction, however, is of great importance, both in morbid anatomy and practical medicine, the more so, as both these affections may exist simultaneously In proof of this I may refer to the controversy that has for some time existed respecting the condition of the mucous membrane of the intestines in fever

I am far from denying the influence of irritation, ulceration, aphthae, and consequent inflammation of the intestinal tunics in continued fevers, and, although they have been more or less noticed and appreciated in all ages, M Broussais has truly benefited his profession by calling the attention of practitioners more particularly to them, and by showing the injurious error of former periods in withholding the employment of general and local bleedings in fevers But we should fall into as great, although an opposite, error, if we concluded that all continued fevers depended on the intestinal irritation that accompanied them, and that every kind of redness observable in them after death indicates a disorder requiring venesection for its treatment The mucous membrane of the stomach and bowel is naturally pale only in persons of pale skins, its degree of colour may be judged of by that of the lips, mouth, arms and vulva, in different individuals No one will set down the livid gums of a dropsical or scorbutic patient, or the swelling and blueness of his hands and feet, to inflammation, or think of treating these affections by blood-letting Now, in many cases, I conceive, the redness of the mucous coat of the intestines has much more relation to this passive congestion than to inflammation If, then, such appearances (as is most probable) only took place in such subjects, at the same time as the lividity of the face and of the dependent parts of the body—that is to say, some days or hours before death, it would be absurd to look to such condition of parts for the cause of the fever,—more especially, as we often find, in such cases, traces of as great or greater disorder in almost every texture of the body For example —the skin is dry and harsh, the lips, gums, and lining membrane of the mouth are swollen, soft and chopped, the membranes of the brain are gorged with blood and containing serum, the lungs are charged with

a sero-sanguinolent fluid, the mucous membrane of the bronchia is swollen and of a violet hue, the heart is flaccid, livid and soft, the blood fluid and imperfectly coagulable, the lining membrane of the arteries or veins livid as if stained by blood, the muscles *fishy* (poisseux), the spleen enlarged, the capillaries of almost every organ, and of the surface, gorged with blood, and lastly, the intestines are in the same state, and their lining membrane livid, ulcerated and thickened in diverse places—Now, to which of these affections shall we attribute the disease? All are posterior—often many days—to the fever Is it not, therefore, more rational to consider, that none of these local lesions are the cause, but that, as in smallpox and measles, some unknown cause, acting generally on the system, had produced both the fever and the local affections—whether active or passive—which accompany or follow it?

In the very case where there exists simultaneously aphthae and exulcerations in the intestinal tunics, and redness, lividity, and capillary congestion of the mucous membrane, we ought to conclude, from analogy, that the two former states are the result of inflammation, active or passive,—and the three latter the result of debility of the circulation in the capillaries, that the first may require venesection, but that this very means, carried to too great lengths, may give rise to, or increase the last, by increasing the general debility The haematemesis and blood fluxes which occur sometimes in continued fevers ought rather, in my opinion, to be attributed to purely passive congestions of the capillaries, than to inflammation In the instance just mentioned we find the whole of the intestinal tunics in the affected part tinged with blood, and softened, without any notable increase of thickness of the part, while inflammation of every mucous membrane uniformly increases both the thickness and density of the part We may farther add the fine observation of Bichat, that, of any morbid affection, inflammation has the least tendency to propagate itself by contiguity, especially in membranous parts Peritonitis and dysentery leave untouched the muscular coat of the intestine, but the lividity consequent on fatal fevers often extends to the whole three tunics

OF MALFORMATION OF THE HEART

There exist two varieties of unnatural communication between the cavities of the heart, viz the perforation of the septum of the ventricles, and the continued patency of the foramen of Botallus The first variety is very rare, there being not more than five or six instances of it on record In all these the unnatural aperture was smooth, evidently very ancient, if not congenital The continued patency of the foramen of Botallus is much more common Sometimes this is produced by the imperfect union of the two plates of the foetal valve, so that a probe, or even a female sound, can be passed obliquely from one auricle to the other This condition of parts is

*Book Third, Chapter 1 Section Seventeenth See footnote on page 332 —F A W 1940

not very rare, and does not appear to be productive of any kind of inconvenience. In other cases we find the foramen continue constantly open so as to admit the finger. I have myself seen it sufficiently large to receive the thumb. It is commonly believed that this species of malformation is always congenital, but from some cases which I have met with, I am disposed to believe that such a perforation may be produced by an accident, or, at least, when such a condition of parts exists as above described, that a blow, fall, or violent exertion, may cause the dilatation of the oblique opening, and its progressive enlargement. The history of several cases on record, especially of some of M. Corvisart's, would seem to countenance this opinion, since, in several of these, the individuals had enjoyed good health, without any symptom of diseased heart, until they had experienced some of the accidental causes above mentioned.

I do not know that any of these unnatural communications have existed without consequent thickening and dilatation of either the whole, or part of the heart, especially the right side. The symptoms of the latter affection are, consequently, combined with those of the former. These are principally the four following: 1, a great sensibility to the impression of cold, 2, frequent faintings, 3, the respiration more constantly impeded than in most other diseases of the heart, and 4, a violet or blueish colour of the skin much more extensive than that in any other disease, and, sometimes, even general. This last symptom has been named by several authors *the blue jaundice*, or *the blue disease*. On the other hand, all the above mentioned symptoms have been found to exist in subjects who had no other malformation than the continued patency of the foramen of Botallus, and still more so in those cases where the pulmonary artery was found to originate in the left ventricle, and the aorta in the right, or where the latter has opened at once into both ventricles. In some diseases of the lungs, especially emphysema, the blue colour of the skin is sometimes quite as intense and as extensive as in the case of malformation of the heart. On the other hand, the foramen of Botallus has been found dilated very considerably, without there being present any degree of lividity except on the face and extremities. The case of dilatation noticed by myself, above mentioned, was of this sort.

OF DISPLACEMENT OF THE HEART

The heart, although retained in its place by the diaphragm, large vessels, and peculiar structure of the mediastinum, and, still more, by the constant state of plenitude of the chest, may, nevertheless, in certain cases, be thrown to the right or left by a solid, liquid, or aeriform effusion into either sac of the pleura, by extensive tumours in the lungs, and, as we have already seen, by emphysema of this organ. In like manner, a tumour in the superior mediastinum or a large aneurism of the arch of the aorta, may press

*Book Third Chapter 1 Section Eighteenth See footnote on page 332—F A W 1940

it downwards, so that that part of the diaphragm on which it reposes shall project into the abdomen Sometimes even this depression has taken place without any visible cause, in which case the affection has been named by some authors *prolapsus* of the heart

These various kinds of displacement produce no perceptible inconvenience when they exist in a slight degree, when more marked, they may produce bad effects, but in this case, they are themselves consequences of lesions much more serious

*CHANGES PRODUCED BY DISEASES OF THE HEART IN THE TEXTURE OF OTHER ORGANS**

On examining the bodies of persons who have fallen victims to organic affections of the heart, besides the organic lesion and the serous effusions which almost always accompany it, we find all the marks of congestion of blood in the internal capillaries The mucous membranes, especially those of the stomach and intestines, are of a red or violet tint, and the liver, lungs, and capillaries situated beneath the serous, mucous and cutaneous tissues, are gorged with blood The augmented colour of the mucous membranes varies much in degree and extent Sometimes it is observed only here and there, under the form of small points or specks, disseminated over the surface of the membrane at other times it occupies the whole extent of the surface, and has the appearance of being attended by some swelling of the part These two latter appearances are sometimes so considerable, that, if we looked to them merely, without examining the condition of the heart, and without reference to the history of the patient (who had been found capable of taking into his stomach wine and other stimulant matters without experiencing any pain, even up to the period of his death), we might be tempted to believe that the fatal disease had been a violent inflammation of the stomach and bowels In fact, the degree of redness of the membranes observed after diseases of the heart, is often much more intense and extensive than is found after true inflammation of these parts, as, for example, in dysentery, a fact, among many others, sufficiently proving the insufficiency of mere redness to characterise inflammation of the mucous membrane of the intestines

Lancisi and Senac, after Hildanus, consider gangrene of the limbs, as a consequence of disease of the heart and large vessels The late M Giraud was of the same opinion, and, since his time, many practitioners have considered the gangrene of old persons as usually caused by ossification of the arteries M Corvisart justly doubts whether, in such cases, there is anything else but mere coincidence of independent diseases, and I think that the single circumstance of the rarity of the spontaneous gangrene of the limbs, compared with the frequency of disease of the heart and ossification of the arteries, is sufficient to render the thing quite improbable This is

*Book Third Chapter 1 Section Nineteenth See footnote on page 332 —F A W 1940

equally the case with the notion of Testa, that ophthalmia, and sometimes the loss of the eye, may be ranged among the consequences of diseases of the heart.*

OF THE CAUSES OF DISEASES OF THE HEART†

The causes of diseases of the heart are, like the diseases themselves, various in their nature. Ossifications are the result of some aberration of the process of assimilation which is not easily understood. I have already stated my opinion respecting the origin of the excrescences on the valves. The dilatation and thickening of the ventricles, diseases of much greater frequency, also may arise from numerous causes, but these are in general more easily traced to their effects, than the former. All diseases which give rise to severe and long-continued dyspnoea produce, almost necessarily, hypertrophia or dilatation of the heart, through the constant efforts the organ is called on to perform, in order to propel the blood into the lungs against the resistance opposed to it by the cause of dyspnoea. It is in this manner that phthisis pulmonalis, empyema, chronic peripneumony, and emphysema of the lungs, act in producing disease of the heart, and that those kinds of exercise which require great exertion, and thereby impede respiration, come to be the most common remote causes of these complaints.

On the other hand, it is found that diseases of the heart, on the same principle of mutual influence, give rise to several diseases of the lungs. They are thus among the most frequent causes of oedema of the lungs and haemoptysis. When, however, diseases of the heart are found to coexist with chronic pleurisy, phthisis, emphysema, and, in general, with chronic disease of the lungs, it will usually be found, on close examination, that the latter are the primary diseases. It follows from these, and other facts noticed under the head of Emphysema and Pulmonary Catarrh, that a *neglected Cold* is frequently the original cause of the most severe diseases of the heart.

To all these causes must be added the congenital disproportion between the size of the heart and the diameter of the aorta. M. Corvisart has, perhaps, gone too far in asserting that there can be no dilatation of the heart without the previous existence of a disproportion of this kind, or of a contraction, or some similar obstruction of the circulation, at a greater or less distance from the heart; it is, however, true, that it is very common to find an aorta of small diameter in cases of hypertrophia or dilatation of the heart. Still, this is not always the case, and however rational such a cause may be, we can readily conceive many others. We know that the energetic and reiterated action of all muscles notably increases their size, as in the case of those of the right arm of the fencer, the shoulder of the porter, and the hands of most artisans. On the same principle we

*Delle Malattie del Cuore. Bologna, 1810.

†Book Third, Chapter I, Section Twentieth. See footnote on page 332.—F. A. W., 1940.

must admit that even nervous palpitations, or such as originate from moral causes, may, by frequent recurrence, produce a true enlargement of the heart

There is yet another congenital cause of disease of the heart, which appears to me to be of greater frequency than the small caliber of the aorta, above mentioned,—I allude to a disproportionate thickness of one or both sides of the heart I am satisfied that in a great many persons the parietes of one or both sides of the heart are either too thick or too thin from birth In such cases there can be no doubt that the usual exciting causes will be more apt to produce formal disease of the heart than in individuals in whom this disproportion does not exist

CHAPTER II DISEASES OF THE PERICARDIUM

Of Pericarditis, or Inflammation of the Pericardium

Pericarditis is inflammation of the serous membrane which lines the fibrous sac of the pericardium, the heart, and large vessels It may be either acute or chronic This inflammation, like that of all membranes of the same kind, is marked by redness, more or less deep, a concrete albuminous exhalation and a sero-purulent effusion The redness is almost always but slight in the acute disease When it exists, it is for the most part only partially It is most commonly punctuated, and looks as if the surface of the membrane was covered, here and there, with little specks of blood very close to each other I have never perceived that this redness was accompanied by any thickening of the part In some cases, wherein, to judge by the thickness of the false membranes, the inflammation appears to have been very great, no redness whatever can be discovered on the serous membrane, on removal of the membranous exudation This concrete albuminous exudation commonly invests the whole surface of the pericardium, as well on the heart and large vessels, as on the sac It rarely presents the appearance of an equable membranous layer, like the false membranes of pleurisy, on the contrary, its surface is most frequently marked by a great number of rough and irregular prominences Sometimes the knobbed appearance of this exudation is very like what would result from the sudden separation of two pieces of slab joined by a pretty thick layer of butter, at other times, it is more like the internal surface of the second stomach of the calf, an observation made, in one case, by M Corvisart In certain cases this aspect of the false membrane has given rise to a singular error, having been mistaken for a variolous eruption in subjects dead of the small-pox The consistence of this exudation is usually greater than that of the false membranes

*Book Third Chapter 2, Section First See footnote on page 332—F A W *1940*

of pleurisy, it is also thicker, and more firmly adherent to the subjacent parts, its colour is, however, the same, being of a pale yellow analogous to that of pus

The serum effused in inflammation of the pericardium is limpid, of a pale yellow colour, or slightly brownish It contains few fragments of semi-concrete albumen, at least, it very rarely contains enough of these to give it a milky and turbid character The quantity of this effusion is usually considerable in the commencement of the disease, often as much as a pound M Corvisart found it, in one case, to amount to four pounds It would seem that the quantity of effused serum diminishes quickly, as soon as the violence of the inflammation begins to subside, as we usually find the proportion of serum and of albuminous exudation nearly equal, while in pleurisy and peritonitis the serum is commonly from twenty to fifty times greater than that of the extravasated lymph Very commonly even, in very violent cases, we find no effused serum, and only a thick and highly concrete albumen filling the whole cavity of the pericardium, and uniting the heart and large vessels to the exterior or loose portion of this membrane In this case we may suppose that the effused serum has been quickly absorbed, and the two layers of false membrane cemented together, although it is not impossible that, in some cases, the more solid exudation may be the only one We have seen that the same thing occasionally takes place in certain partial and sub-acute inflammations of the pleura, and several observations have led me to believe that the cartilaginous patches that sometimes are met with on the exterior of the lungs are produced in the same manner

When the disease terminates favourably, the pseudo-membranous exudation, after a certain time, is converted into cellular substance, or rather into laminae of the same nature as the serous membranes, that is to say, the laminae are double, the exterior surface being exhalent, and the interior cellular, or adherent, and containing the vessels distributed to the part Sometimes these laminae are long, sometimes so short that the pericardium seems intimately adherent to the heart

Before the conversion of false membranes into cellular tissue was well understood, the adhesion of the pericardium to the heart was regarded by divers authors as a cause of various and serious complaints Lancisi and Vieussens considered it as constantly causing palpitation, Meckel, as rendering the pulse habitually small, and Sénac, as productive of frequent faintings Even M Corvisart himself has fallen into some mistakes on this head He admits three species of adhesions,—all of which I have just described as mere varieties or stages of the same affection These are, first, a semiconcrete albuminous adhesion, which is the only one recognised by him as the consequence of pericarditis, second, the very intimate or close cellular adhesion, deemed an effect of gouty or rheumatic affections, and third, the extended or long cellular adhesion,

the cause of which is not assigned by him * M Corvisart is further of opinion that no person can live, and preserve a good state of health, who is affected with a complete and close adhesion of the pericardium to the heart, or of the lungs to the pleura

I have, however, met with many cases where this condition of parts was found after death, in which no disorder of the respiration or circulation existed during life A case adduced by M Corvisart in support of his opinion (Op cit p 34) appears to me rather conclusive against it, inasmuch as the appearances on dissection showed sufficient lesions in other organs to account for the symptoms referred by him to the adhesions between the heart and pericardium

Sometimes, though rarely, the inflammation is confined to a part only—sometimes a very small part—of the pericardium These partial inflammations are in proportion to the general, in point of frequency, hardly as one to ten Their anatomical characters are precisely the same, only that the albuminous exudation is in them confined to the inflamed part The serous effusion is sometimes as abundant as in the general disease, more commonly, however it is less The inflammation in this case almost always terminates in being cured, by the transformation of the pseudo-membranous exudation into long serous laminae, scarcely ever are these partial inflammations followed by the intimate adhesion of the parts

We frequently find on the surface of the heart opaque white patches, sometimes as large as the palm of the hand, more commonly one-half or one-third this size, and often very small They are nearly of the thickness of the nail, and have a degree of consistence equal to that of the membranes composed of condensed cellular substance, as, for instance, the exterior membrane of the lymphatic glands They adhere so closely to the parts on which they lie, that it is difficult to ascertain, even by dissection, whether they are situated above or beneath the fine membrane covering the heart and great vessels M Corvisart is of opinion that they are beneath it I have, however, ascertained the incorrectness of this opinion, as I have several times been able to remove the patches, leaving the serous membrane of the pericardium still untouched

Are these patches the effect of partial pericarditis and the consequent conversion of the effused lymph into a condensed membranous cellular tissue? M Corvisart considers them as produced without previous inflammation, and seated, as I have already said, beneath the serous surface of the pericardium Both these notions are, I think, inadmissible, inasmuch as there exists no example of an albuminous exudation on the adherent surface of a serous membrane, and as facts without number prove that pseudo-membranous exudations are always the produce of inflammation

*See Treatise on the Heart etc, by M Corvisart

I have lately met with a case which appears to me to throw some light on the question of the origin of these spots In a man dead of peripneumony, I found a thin false membrane, very firm and of a yellowish colour, investing the right auricle and a portion of the ventricle of the same side, all the rest of the pericardium being quite free, only containing in its cavity two or three ounces of a transparent and slightly yellow serum Some parts of the false membrane, particularly on the auricle, were of a whiter colour and firmer than the rest, and exhibited an appearance, almost the same as the white patches above described

Chronic pericarditis is always general occupying the whole internal surface of the serous membrane This is commonly much redder than in the acute disease The redness is formed by the close approximation of minute points, which look as if applied with a pencil Rarely the chronic disease is accompanied by a pseudo-membranous exudation, and when this exists, it is thin, soft, friable, and entirely resembling a layer of very thick pus In every case there exists a more or less copious effusion of a turbid, milky fluid, sometimes having quite a puriform character I am led to believe that the close adhesion of the pericardium to the heart, is commonly the consequence of the absorption of this fluid, and that the adhesion by the long laminae is the product of the acute disease In one case I found a close and general adhesion of the pericardium to the heart and large vessels, by means of a false fibro-cartilaginous membrane, in every respect like those of the pleura

From one case, cited by M Corvisart, I am led to believe, that there may occasionally arise, subsequently to chronic inflammation of the pericardium, a tuberculous eruption similar to those frequently formed in the false membranes of the pleura and peritoneum "The portion of the pericardium," he says, "which invests the heart, was of a greyish colour, thickened, unequal, wrinkled, crisp, and containing granulations of which the summit seemed ulcerated " I am the rather led to consider these *granulations* as tubercles, because in the same subject "both lungs, although crepitous, were *granular* throughout "

In many cases of pericarditis, especially in the chronic disease, the muscular substance of the heart has lost its colour and become whitish This loss of colour is sometimes attended by a notable degree of softening, and, at other times, the consistence is natural Most writers have regarded this loss of colour as a mark of the inflammation of the heart itself, and most of the examples recorded of Carditis are merely cases of inflammation of the pericardium accompanied by this loss of colour A great number of those collected by M Corvisart are of this kind For my own part I am disposed to doubt the correctness of the opinion that refers this loss of colour to inflammation We can never be sure of the existence of inflammation in a muscular organ unless we find a deposition of pus among its fibres

PERICARDITIS

1 *Acute Pericarditis* There are few diseases attended by more variable symptoms, or of more difficult diagnosis, than this Sometimes it appears with all the symptoms of a very violent disease of the chest, at other times it proves fatal without leading us, in the least, to suspect its existence Again, we find cases marked by all the symptoms usually attributed by nosologists to this disease, and in the subjects of which, after death we discover no traces of its existence The same difficulty is acknowledged, or at least encountered, by most practitioners Corvisart attributes the difficulty to the circumstance of pericarditis being almost "always complicated with pleurisy, peripneumony, or some other disease of the chest, which masks its peculiar symptoms " These complications, which are very common, must, unquestionably, have this effect where they exist, I must, however, confess, that the most completely latent affections of this kind that I have met with, were in subjects whose thoracic viscera were, in every other respect, quite sound, and who had died of disease of the abdomen These facts seem to prove that inflammation of the pericardium is sometimes a local affection of little violence, and of very inconsiderable influence on the general system or even on the circulation, while, in other cases, it is accompanied by an acute fever, and by such violent disorder of almost all the functions, as to compromise the life of the patient

M Corvisart is likewise of opinion that it is when the disease is very acute, that the symptoms are very obscure "Its invasion," he says, "is sudden, its progress rapid, its termination almost instantaneous " When it exists in a less violent degree, but still acute, he thinks that it can be recognised by the following symptoms viz sense of heat in the region of the heart, great difficulty of respiration, greater colour of the left cheek than the right, pulse at first frequent, hard and rarely irregular,—becoming about the third or fourth day, small, hard, contracted and often irregular, great anxiety, slight palpitations, partial faintings, *peculiar* change of features, and (towards the close of the disease) total or partial cessation of the local pain

These symptoms are certainly sometimes present in pericarditis, but each, or all of them may be absent, and some of them are very rare I have never observed the increased colour of the cheek, have rarely heard complaints of local heat or pain, and, in place of the progressive increase of irregularity in the pulse (as described by M Corvisart), I have uniformly found this irregularly intermitting, wiry, and almost insensible, from the very commencement of the disease

I must admit that the stethoscope scarcely furnishes us with any more certain signs of this disease The following appear to me to be the most common symptoms of the inflammation of the pericardium, when not latent, the contraction of the ventricles yields a greater shock, and sometimes a

more marked sound, than usual, and, at intervals, feebler and shorter pulsations are perceived, which correspond with intermissions of the pulse, the smallness of which contrasts remarkably with the strength of the heart's pulsation. When these symptoms come on suddenly in a person who had never been affected with disease of the heart, there is great probability of their being the consequence of this disease. In addition, it is further common for the patient to have much dyspnoea and very great anxiety, and to suffer syncope on taking a few steps, or on moving suddenly in his bed.

2. *Chronic Pericarditis.* The signs of this variety are still more uncertain than those of the acute disease. I have attended several cases which I considered, throughout their whole course, as chronic inflammations of the pericardium, but which almost all were cured. In two or three cases only have I been able to verify the correctness of my diagnosis by examination after death, whilst very frequently I have found the pericardium full of pus and in a true state of chronic inflammation, without having been at all led to suspect such an affection. In the cases which have occurred within the last three years, I have found the symptoms to be precisely the same as in the acute disease, only less violent. From one to two years has elapsed before a cure has taken place, and when this has been effected the action of the heart and pulse has become natural and regular.

*OF HYDRO-PERICARDIUM, OR WATER IN THE PERICARDIUM**

It is extremely common to find a greater or less quantity of serum in the pericardium, most frequently this does not exceed a few ounces, and can rarely be considered as idiopathic. Most commonly it can only be regarded as taking place in articulo mortis. When there exists a general dropsical diathesis, we occasionally find some water in the pericardium, but, in general, it contains less than the other serous cavities. In the idiopathic hydro-pericardium, on the contrary, the pericardium is commonly the only membrane which contains serous effusion.

The effused serum is sometimes colourless, but more commonly it is yellowish, brownish or reddish, although still perfectly limpid, and without any admixture of flakes of lymph, rarely it is sanguinolent. It is variable in amount. Most frequently it does not exceed one or two pounds, but it has been found in much greater quantity than this. M. Corvisart records an instance wherein eight pounds were found. This effusion is attended by no change in the heart or its coverings. Some authors have, indeed, stated the heart to have been macerated (macéré) in such cases, but I am disposed to consider such statements as the result of imperfect observation and incorrect description. Very frequently before opening a pericardium partially filled with serum I have distinctly observed an accumulation of air in the cavity. I have seen this occupy a space the size of the

*Book Third. Chapter 2. Section Second. See footnote on page 332.—F. A. W. 1919.

fist, and when as large as this, a distinct hissing sound is perceived in puncturing the pericardium In place of this continuous mass of air, we more commonly observe a great quantity of small air-bubbles on the surface of the liquid I am inclined to think that I have found air in the pericardium in cases where there was no serum, but I am not quite assured of the correctness of my observation At all events, this case of simple *Pneumo-Pericardium* is extremely rare, while the other variety, just described, is by no means so

Authors vary respecting the symptoms of this affection [hydropericardium] Lancisi states the principal to be a sensation of an enormous weight in the region of the heart Reimann and Saxonia assure us that the patient feels his heart swimming in water Sénac says he has seen the fluctuation of the fluid between the third, fourth, and fifth ribs M Corvisart says he has perceived this fluctuation by the touch, and adds the following as marks of the affection —*sense of weight in the region of the* heart, inferior resonance on percussion, pulsation of the heart irregular and obscure, and felt over a large space and with variable intensity in the same and different points of this space, pulse small, frequent and irregular, threatened suffocation on lying in the horizontal posture, frequent syncope, but rarely palpitation, oedema To these symptoms I may apply the same remarks as to those of pericarditis they may exist, in greater or less number, with or without hydro-pericardium I am unable to say, from experience, how far, and in what respect, the cylinder will assist the diagnosis of this disease

*OF ACCIDENTAL PRODUCTIONS IN THE PERICARDIUM**

Various species of accidental productions have been found between the pericardium properly so-called, and the pleura, also, between it and the internal and serous membrane, and, lastly, between the serous membrane and the heart In the Sepulchretum of Bonetus and other collections of cases, we find examples of what appear to be tubercles, cancerous tumours, or cysts, in the different situations just mentioned But the imperfect knowledge of membranes before the time of Bichat, and the general confusion of all accidental productions under the names of Scirrhus, Carcinoma, Atheroma, etc renders it impossible to ascertain precisely either the nature or site of such morbid growths I have already noticed the fatty productions, in the form of a cock's comb, developed occasionally between the pleura and fibrous membrane of the pericardium Twice or thrice I have found tubercles in the same situation, in subjects which exhibited a great number of these bodies in the lungs and elsewhere I have also seen a tubercle situated at the point of the origin of the pulmonary artery and beneath the serous membrane of the pericardium

*Book Third, Chapter 2 Section Third See footnote on page 332 —F A W, 1940

Once only have I met with an instance of ossification between the layers of the pericardium As this case was remarkable both for its extent and the effects produced by it, I shall here briefly detail it

Case 45 A man, aged 65 years, had led an intemperate life, but had, nevertheless, enjoyed good health until his fiftieth year At this time he appears to have had an attack of pleurisy of short duration, but which was followed by oedema of the lower extremities and subsequently by anasarca of other parts, and by dyspnoea and breathlessness on ascending an elevation, or using any degree of exercise When he came into hospital, in the end of spring, the dropsical symptoms continued and the lips were swollen and violet The pulsations of the heart were unequal, irregular, and very distinct, though perceptible over a very small extent of the chest The pulse was feeble, small, soft, unequal, intermittent and irregular There was no cough, but copious expectoration The thorax sounded well superiorly, but badly on the lower parts

The patient could lie in any posture, slept well, even without having his head raised, and had no sudden startings from sleep —He died in the course of a few months, the dropsical swellings and dyspnoea having much increased The brain, lungs, and abdominal viscera were found in a sound state The heart was enlarged, and adhered throughout to the pericardium, by means of very close cellular attachments On first touching it, it seemed to be quite inclosed in a bony case, situated beneath the fibrous membrane of the pericardium, but on further examination this incrustation was found to be incomplete Around the base of the ventricles there was a zone or band, partly bony and partly cartilaginous, of from one to two fingers' breadth, of unequal thickness, flattened, yet somewhat rough on its surface This band projected into the angle between the ventricles and auricles, and extended along the interventricular septum on both sides, to near the apex of the heart The whole of this production was contained between the fibrous membrane of the pericardium and the serous membrane which lines it internally The auricles were enlarged so that each might have contained a large egg One of the mitral valves contained an ossified point of the size and shape of a French bean

OF ANEURISM OF THE AORTA*†

In the following observations I shall adhere to the ancient distinction of *true* and *false* Aneurisms,—the former comprehending dilatation without rupture of any of the arterial coats, the latter with rupture of some of these

True aneurism of the ascending portion and arch of the aorta is very uncommon The dilatation usually extends from the origin of the artery to the point were it begins to descend This dilatation rarely proceeds so

*This chapter is more abridged than the others —*Trans* [Forbes]

†Book Third Chapter 3 See footnote on page 332 —F A W 1940

far as to produce very serious symptoms, the extreme point of dilatation of the artery not being wider than from two to three fingers' breadth The convexity of the arch and anterior part of the artery appear to yield more than the other parts of the vessel When the dilatation exists in the descending aorta, it assumes the form of an ovoid tumour, gradually terminating, at each extremity, in the undilated artery It is not uncommon to find several dilatations of this kind in the same artery Sometimes we find the whole tract of the aorta dilated to double its natural size

Dilatation in the arch of the aorta, in the degree above described, is very common, but this is not usually named *aneurism* unless it arrives at a considerably greater extent These sometimes are very large M Corvisart records one double the size of the heart, and I have seen them of the size of the head of a full-grown foetus When the *true aneurism* acquires a certain size, the inner coat often is ruptured and a *false* aneurism ensues The true aneurism is commonly accompanied with a morbid degeneration of the internal tunic of the artery It exhibits spots of a bright red, slight cracks, and a great number of small ossified points These latter are usually considered as contained in the substance of the inner coat, but they are, in truth, situated between it and the middle coat

The false aneurism of the aorta, consequent to the true, is rarer than the simple dilatation of that artery, but it is much more common than that greater degree of simple dilatation which alone usually claims the name of *aneurism*

The false aneurism is most common in the ascending, and the true in the descending aorta I have never met with any other species of false aneurism in the ascending aorta, or its arch, but that consequent to the true, or simple dilatation of the part In the descending aorta, however, false aneurism often takes place without any previous dilatation The opinion at present current in the Parisian schools, viz that in aneurism the internal coat remains entire and protrudes, in the form of a hernia, through the ruptured fibrinous tunic, is more untenable, as a general position, than that of Scarpa, who maintains the rupture of the two internal tunics in every case of the disease Both these opinions are true in certain cases, but not in all

Aneurisms of the aorta produce various effects on the adjacent organs, according to their volume and position Simple dilatation, when in a moderate degree, hardly produces any effect, but the most inconsiderable false aneurisms may give rise to very serious disorder The first and most common of these effects is compression acting on the heart and lungs When the aneurism is in contact with the lungs, it most commonly merely compresses them, sometimes, however, the substance of these organs gives way, and the aneurism, when it bursts, pours its blood directly into the air-cells Frequently the aneurism compresses the trachea, or one of the two

bronchial trunks, flattens, and eventually destroys a part of them, and death ensues by a species of haemoptysis from the rupture of the tumour. The same thing occasionally happens with the oesophagus, but not so frequently. I have only met with three instances of death from this cause. The ordinary effect of these aneurisms on the heart, is to displace it more or less, downwards or to one side. Sometimes the aneurism bursts into the pericardium (see Morgagni and Scarpa), but I have never met with an example of this. A case is on record of an aneurism of this kind bursting into the pulmonary artery. The left cavity of the pleura is, by far, the most frequent situation for the rupture of these aneurisms. I have met with one case where the aneurism compressed and destroyed the thoracic duct, and M. Corvisart notices a fatal case of compression of the superior vena cava from the same cause. The most remarkable local effects of aneurisms of the aorta, are those on the vertebral column. They often destroy this to a very great depth. This destruction is entirely the work of interstitial absorption, there never being any mark of caries. On the side next the vertebrae the sac is completely destroyed, and the circulating blood is bounded by naked bone.

Aneurisms of the ascending aorta destroy, in like manner, the sternum by their pressure, so that they are at length covered merely by the skin. I have met with two or three tumours of this sort so large that they could not be completely covered by both hands. The aneurisms of the arch of the aorta, and of the arteria innominata, sometimes project, in like manner, at the top of the sternum or above it, or under the cartilages of the first false ribs of the right side. It is not always the largest aneurisms that most readily make their way externally. Sometimes those of the size of an egg produce this effect, whilst, occasionally, those of the size of the head of a full-grown foetus remain quite covered and are even compressed by the sternum.

ANEURISM OF THE AORTA

There are few diseases so insidious as this. It cannot be certainly known till it shows itself externally. It can hardly be suspected, even when it compresses some important organ and greatly deranges its functions. When it produces neither of these effects, the first indication of its existence is often the death of the individual as instantaneously as if by a pistol-bullet. I have known men cut off in this manner, who were believed to be in the most perfect health, and who had not complained of the slightest indisposition. We must, therefore, admit that aneurism of the aorta has no symptoms peculiar to it: all those noticed by authors, and especially by M. Corvisart, being indicative merely of the change or compression of adjoining organs. This will be evident by the enumeration of the principal of these, viz. oppression of the chest—dissimilarity

*Bulletin de la Faculté de Méd. 1819.—*Trans.*

of the pulse in both arms,—a wheezing or rushing at the top of the sternum, perceptible by the hand,—obscure sound on percussion,—rattling in the throat, and dragging downwards of the larynx, when the tumour compresses the trachea, etc. After what has been said of the symptoms of other diseases of the chest, I need not remark how very equivocal all these are. In the present state of our knowledge there certainly exists no certain means of ascertaining the existence of this disease until it shows itself externally. And hitherto, my experience has been insufficient to enable me to say how far this difficulty is likely to be removed by the use of the stethoscope. Since my employment of this instrument I have met only with a dozen cases of what I conceived to be aneurisms of the aorta. Most of these left the hospital after obtaining relief by blood-letting and proper diet. In two instances of moderate dilatation of the arch, I was enabled to verify by dissection my previous diagnosis afforded by the cylinder, and in a third, which showed itself externally, I was enabled to verify still further the diagnostic indications. In this last case, I found the pulsations of the tumour perfectly isochronous with the pulse at the wrist; they gave, at the same time, a much greater impulse and louder sound than the mere contraction of the ventricles, and the contraction of the auricles was not at all perceptible. This pulsation, which I shall call *simple*, in opposition to that of the heart, which is *double* (including the alternate contraction of auricles and ventricles), was distinctly perceptible between the right scapula and the spine. In some cases, this *simple* pulsation and greater impulse may indicate the disease, but I must confess that I have myself been deceived in three cases notwithstanding these indications. I would, therefore, say that even this *simple* pulsation will not assist us in distinguishing aneurisms of the arch or ascending aorta from dilatation of the ventricles.

Another sign, however, will still remain, though less marked than the *simple* pulsation above mentioned. It is this. If we find under the sternum, or below the right clavicle, the impulse of the circulatory organ isochronous with the pulse, and perceptibly greater than that of the ventricles examined in the region of the heart, we have reason to suspect dilatation of the ascending aorta, or arch,—the more so, as it is extremely rare to feel the impulse of the organ of circulation beyond the region of the heart, even in cases of the most marked hypertrophia.

The whole of my experience on this subject leads me to the following conclusions: first, in several cases aneurisms of the ascending aorta can be ascertained by the cylinder; second, in other cases, it requires the greatest attention to distinguish their pulsation from that of the heart; third, aneurisms of the pectoral aorta can be recognised, more especially when they have produced injury of the vertebral; and fourth, all of them will be often mistaken, because nothing will lead to the examination of the chest, and because there will often be no sign whatever of ill-health.

1825

CALEB HILLIER PARRY

DESCRIPTION OF THE CIRCULATORY PHENOMENA IN EXOPHTHALMIC GOITER

CALEB HILLIER PARRY

Engraving by Philip Audinet from a miniature sketch by John Hay Bell, 1804

(Courtesy Annals of Medical History)

CALEB HILLIER PARRY

(1755-1822)

CALEB HILLIER PARRY was born on October 21, 1755, in Cirencester in Gloucestershire, the eldest son of a family of ten children. His father, Joshua Parry, was a nonconformist minister.

Young Caleb attended grammar school in Cirencester, where he made the acquaintance of Edward Jenner, a classmate. Jenner and he subsequently became life-long friends. In 1770, Parry was sent away to school to the Dissenters' Academy at Warrington in Lancashire.

Parry became a student of medicine in 1773, at the University of Edinburgh. At that time the medical department of that institution was dominated by William Cullen, the great teacher.[1] After spending two years at Edinburgh, Parry went to London. In London he lived for a time with Dr Thomas Denman (1733-1815), obstetrician to the Middlesex Hospital. At London, Parry probably gained some valuable experience. Two years later, 1777, he returned to the University of Edinburgh. He received the degree of Doctor of Medicine in 1778. His thesis was entitled "De rabie contagiosa vulgo canina." Thirty-six years later, in 1814, Parry again wrote on rabies and dedicated the new work to Jenner.

In October, 1778, Parry was married to the daughter of John Rigby of Manchester. Their honeymoon was spent on the Continent, and after the trip they returned to England and settled in Bath in 1779. There Parry spent the rest of his life.

Shortly after choosing Bath for his residence he became physician to the Puerperal Charity Hospital. Later, in 1789, he became physician to the Casualty Hospital.

At Bath, at the beginning of his career, Parry, like many newly graduated physicians of that era, found that the practice of medicine did not demand all his time. He therefore devoted his leisure to the collection of fossils, and in this manner acquired a large collection. In 1781 he published his "Proposals for a History of Fossils of Gloucestershire."

In 1788 Parry became a licentiate of the Royal College of Physicians of London. By this time he enjoyed a satisfactory practice. Among his patients were the German astronomer and philosopher, Sir William Herschel, Senior (1738-1822), and Admiral Lord George Rodney (1718-1792). Parry named his third son after the distinguished naval officer. This son died in infancy in 1786. Another son died at the age of twenty-one. His most famous son, William Edward Parry, was destined to become a rear admiral, after having gained renown as a famous Arctic explorer. His eldest son, Charles Henry, became the first physician to the Royal United Hospital in Bath, which was founded in 1826 when the Casualty Hospital merged with the Bath City Infirmary.

Parry, in 1789, presented a paper before the Medical Society of London entitled "On the Effects of Compression of the Arteries in Various Diseases, and Particularly Those of the Head, with Hints towards a New Mode of Treating Nervous Disorders." This was published in 1792. Therein he observed the beneficial effects,

[1] See page 227, biographic sketch of William Withering.

in the presence of fits, of compressing the carotid artery of the patient and thus causing a diminution of the blood supply to the brain By applying a tourniquet to the arteries of the limbs, he also observed improvement of patients who had certain diseases of the extremities

Parry published an important study on angina pectoris in 1799 entitled "An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris, illustrated by Dissections"

In 1815 appeared the first volume of Parry's "Elements of Pathology and Therapeutics" He had hoped to publish a second volume but did not live to finish it His son, Charles Henry Parry, republished this work in 1825, including the unfinished second volume

In 1816, Parry published the results of some experimental studies he had conducted on animals It was entitled "An Experimental Inquiry into the Nature, Cause, and Varieties of the Arterial Pulse" Although some of the observations which he records are inaccurate, his conclusion that the pulse wave is caused by the impulse given to the blood by the systole of the left ventricle is in agreement with present knowledge

In October, 1816, Parry was stricken with right hemiplegia and aphasia, which prevented him, unfortunately, from producing any more serious works of a medical nature He devoted his last years to the care of his farm and garden, to reading, and, with his daughter's assistance, to the collecting of miscellaneous items and anecdotes, which were recorded in many volumes

Parry died on March 9, 1822, at his house in Sion Place, Bath He was buried in the Abbey Church at Bath, where a memorial was erected to him by his fellow practitioners

Among Parry's accomplishments of a nonmedical nature was his aid in promoting the wool industry for Great Britain For his many services he was elected an honorary member of the Farming Society of Ireland He was also a member of the Royal Society of London and the Society of Natural History of Gottingen in Germany

In 1825, three years after Parry's death, a collection of his hitherto unpublished medical writings was assembled and published by his son, Charles Henry Parry Of utmost interest was Parry's description of exophthalmic goiter and the accompanying circulatory phenomena We are privileged to reprint this classic description His first case was observed in August, 1786, and thus predated by fourteen years Joseph Flajani's publication in 1800 Of course, Parry's account was made several years before the classic descriptions of Robert Graves and Karl von Basedow

ENLARGEMENT OF THE THYROID GLAND IN CONNECTION WITH ENLARGEMENT OR PALPITATION OF THE HEART*

By

CALEB HILLIER PARRY

CASE 1.—There is one malady which I have in five cases seen coincident with what appeared to be enlargement of the heart, and which, so far as I know, has not been noticed, in that connection, by medical writers. This malady to which I allude is enlargement of the thyroid gland.

The first case of this coincidence which I witnessed was that of Grace B., a married woman, aged thirty-seven, in the month of August, 1786. Six years before this period she caught cold in lying-in, and for a month suffered under a very acute rheumatic fever, subsequently to which, she became subject to more or less palpitation of the heart, very much augmented by bodily exercise, and gradually increasing in force and frequency till my attendance, when it was so vehement, that each systole of the heart shook the whole thorax. Her pulse was 156 in a minute, very full and hard, alike in both wrists, irregular as to strength, and intermitting at least once in six beats. She had no cough, tendency to fainting, or blueness of the skin, but had twice or thrice been seized in the night with a sense of constriction and difficulty of breathing, which was attended with a spitting of a small quantity of blood. She described herself also as having frequent and violent stitches of pain about the lower part of the sternum.

About three months after lying-in, while she was suckling her child, a lump of about the size of a walnut was perceived on the right side of her neck. This continued to enlarge till the period of my attendance, when it occupied both sides of her neck, so as to have reached an enormous size, projecting forwards before the margin of the lower jaw. The part swelled was the thyroid gland. The carotid arteries on each side were greatly distended, the eyes were protruded from their sockets, and the countenance exhibited an appearance of agitation and distress, especially on any muscular exertion, which I have rarely seen equalled. She suffered no pain in her head, but was frequently affected with giddiness.

For three weeks she had experienced a considerable degree of loss of appetite, and thirst, and for a week had oedematous swelling of her legs

*Parry, C. H.: *Collected Works*, London, 1825, Vol. I, pp. 478-480. We reprint from Major, R. H.: *Classic Descriptions of Disease*, Springfield, Ill., 1932, Charles C. Thomas.—I. A. W., 1940.

and thighs, attended with very deficient urine, which was high coloured, and deposited a sediment. Until the commencement of the anasarcous swellings, she had long suffered night sweats, which totally disappeared as the swellings occurred. She was frequently sick in the morning, and often threw up fluid tinged with bile.

CASE 2.—August 22, 1803. Elizabeth S., aged twenty-one, was thrown out of a wheel chair in coming fast down hill, 28th of April last, and very much frightened, though not much hurt. From this time she had been subject to palpitation of the heart, and various nervous affections. About a fortnight after this period she began to observe a swelling of the thyroid gland, which has since varied at different times, so as to be once or twice nearly gone. It is now swelled on both sides, but more especially the right, without pain or soreness on pressure. The pulsation of the carotids is very strong and full on both sides, but evidently in the greatest degree on the right. Menses regular, and bowels uniformly open. She voluntarily tells me that she used to be very subject to headaches, which have ceased ever since the commencement of these swellings. Pulse 96, small, hard, and regular.—Mitt^r Sanguis è Brachio ad ℥x.

Her head was much relieved by the blood-letting, and the swelling of the thyroid gland was evidently diminished.

On the 25th, she was ordered to take thrice a day a teaspoonful of a mixture of Tincture of Digitalis thirty drops, Syrup of Squills an ounce and a half.

August 31. The medicine made her sick on the second day, but she has continued it ever since without the same effect. Her bowels have been regularly purged once or twice a day, but the palpitation of the heart has been frequent, especially on exercise, which much fatigues her. Swelling of the thyroid, and beating of the carotids, much as before. Pulse 96. Mitt^r Sanguis ad ℥x. Pergat in usu Syrupi, 4^ter in die.

September 7. Bowels open. No sickness. Palpitation somewhat better. Swellings nearly as before, that on the right being still the largest, and the pulsation of the carotid on that side the greatest.—Pergat.

September 14. All complaints nearly gone. Bowels open without sickness. Pulse about 72, and slightly irregular as to the force of the strokes. Pulsation of the carotids still too strong. Swellings lessened. Menses adsunt.—Pergat in usu Syrupi.

September 24. Yesterday morning she was seized with giddiness and sickness without vomiting. Bowels open yesterday and frequently today. On the 14th ultimo, she was menstruating, and continued to do so for three or four days, during which the swelling of the thyroid almost disappeared, but has since returned, and the beating of the carotid is very strong. She has at this time some catarrh with sore throat.—Pergat in usu Syrupi.

October 1. The symptoms of catarrh are gone, and the swellings are again very much lessened, though the pulsation of the carotids, especially

the right, is still too strong That of the heart, on exercise, is much diminished Two stools daily, less loose than before —Pergat in usu Syrupi cum Tincturae Digitalis ʒj

Case 3 —Mrs K , aged about fifty, a very thin woman, had for many years laboured under violent and often irregular action of the heart, accompanied with more or less of shortness and difficulty of respiration During several aggravations of this disease I attended her, and found her heart violently palpitating, so as to reach 136 beats in a minute, extending its throbbing both downwards and on the right of the thorax, far beyond the due limits, and swelling in a preternatural degree all the arteries which were capable of being felt, and more especially the carotids The pulse was often unequal both as to frequency and strength The respiration was greatly hurried, and the head was affected with throbbing pains The urine was often defective All muscular exertion aggravated the symptoms, which were occasionally relieved by blood-letting, Squills, Digitalis, and aperients Still, however, much of the malady continued, and I could never perceive that the pulse was reduced below 120 in the minute

Mrs K was also long affected with an extremely large swelling of the thyroid gland, which began at a period, the relation of which to the commencement of the disorder of the heart, she was unable to recollect

My last attendance on her was in June, 1813, on the 24th of which, at eight in the morning, I was called to visit her, and found her in bed Her pulse was 132 in a minute, and very full, hard, and strong, both in the radials and carotids The beating of the heart extended all over the thorax, and even into the right hypochondrium The respiration was 24 in a minute, with grunting expiration, and with no elevation of the diaphragm during inspiration She had occasional cough, with yellowish brown mucous expectoration The thyroideal swellings projected before the carotids, and involved the sterno-mastoid muscles from their lower insertion to nearly two-thirds of their length upwards The carotids were driven somewhat forward, and much enlarged, and the external jugulars were swelled and prominent For about a fortnight she had been affected with an oedematous swelling of her legs, which had gradually increased The abdomen was also tense, but not fluctuating, and she suffered considerable pain about the navel, where there was soreness on pressure The bowels had however been open during the night, with griping The quantity of urine had not exceeded a teacup full in the last forty-eight hours Some medicines were given, which it is needless to specify, as the patient died at five o'clock the next morning A dissection was not permitted

Case 4 —A woman servant, unmarried and about thirty years of age, whom during a space of several months, I had at various times seen labouring under a palpitation of the heart, which always more or less

existed, and was accompanied with a quick and irregular pulse, great hurry in breathing on an exertion, and an extremely strong beating of the carotid arteries, began at length to have enlargement of the thyroid gland, which had not existed more than a fortnight when I last saw her, and which was much increased from the time when it was first noticed.

Case 5.—During my attendance on this patient, I was consulted by a married lady, of about forty years of age, from the North of England, who was supposed to be in consumption. She had in fact a very quick pulse, with great shortness and difficulty of breathing, and frequent cough, attended with copious expectoration. She had also an extremely large swelling of the thyroid gland on each side of the neck, with a considerable dilatation of the carotid arteries. The cough having been removed in about a fortnight by blood-letting, Squills, and Citrate of Potash, which were ordered when she first consulted me, I had an opportunity of discovering, at my second visit, that she was afflicted with a most laborious action of the heart, which, from the extent of the pulsation, seemed much enlarged, and suffered a great aggravation of symptoms from any muscular exertion.

This inordinate action of the heart has been of long duration, and considerably preceded the commencement of the thyroideal swelling.

The patient did not remain at Bath long enough for me to know the result of the disease, which, doubtless, would ultimately prove fatal.

My attendance on the three last patients having occurred at the same time, first suggested to me the notion of some connection between the malady of the heart and the bronchocele. I mentioned that opinion to Mr. G. Norman, surgeon, to whom I shewed the lady last mentioned. Shortly afterwards I expressed the same opinion to Mr. Cruttwell, surgeon, to whom it then occurred that he was attending a patient with a similar coincidence, and that in her the bronchocele succeeded to the affection of the heart.

Case 6.—Anne P., aged about thirty, a married woman, thin, and with a very long neck, who has never had a family, five years ago, at Christmas, when affected with chilblains, for their relief kept her feet in cold water for a quarter of an hour, which made her feet extremely cold. Half an hour afterwards she was seized with a pain about the region of the heart, which was extremely violent, but unaccompanied with cough, fever, or palpitation. Ever since that period she has been subject to attacks of similar pain, which recur frequently. She has also frequent palpitations, which come on more especially after walking or any hurry, though sometimes without any apparent cause whatever. She is often affected also with oppression of breathing, which is sometimes accompanied with globus hystericus, and obliges her to lie high in bed. All pressure about the thorax is uneasy to her; but she lies best on her left side. She is free from cough. At this moment she complains of violent pain on the sternum towards the lower part, which is not sore on pressure. Pulse 112, and weak. Respiration 22. Extremities

cold Skin pale She is sleepy during the day, but sleeps little at night Tongue rather furred Appetite irregular Urine very various as to appearance Menses, since the commencement of the malady, defective

During the palpitation, and indeed at other times, she has long had a violent beating in her head, and a throbbing in her neck This day fortnight she had an unusual degree of this throbbing, accompanied with a great aggravation of a distracting pain in the head, to which she has been subject ever since she began to be ill, and which is always greatly increased by coming out of the air into a warm room During the more violent accessions of this affliction of the head, she cannot bear the least conversation, and feels as if she should die The evening after the last described aggravation, the thyroid gland began to swell at its lower part before, and the swelling has now diffused itself to a considerable degree on each side, without soreness on pressure The beating of the carotids is very strong

1827

ROBERT ADAMS

HEART BLOCK

ROBERT ADAMS

(Courtesy Medical Classics)

ROBERT ADAMS

(1791-1875)

"*that 'Old Guard' of Irish surgery*"

—*The Lancet* (London)

ROBERT ADAMS, who was born in Dublin, who spent most of his life there, and who died in the city of his birth at the ripe old age of eighty-four, was, according to the "Lancet," the last of the old guard of illustrious Irish surgeons of the nineteenth century, the roll commencing with the elder Dease and Peile In the charter granted the Royal College of Surgeons in Ireland by King George IV in 1828, Adams is listed as one of the group to whom the charter was granted And on the roll of the Supplemental Charter bearing the date of January 24, 1844, his name is the eighteenth that occurs Immediately preceding his name on this record are the names of Peile, the eminent lithotomist, Crampton, Kirby, Read, Cusack, Jacob, and William Henry Porter

In 1810 Adams entered the University of Dublin as a student of the liberal arts He studied intermittently at the University for several years receiving the degree of Bachelor of Arts in 1814, the degree of Master of Arts in 1832, and the degree of Doctor of Medicine in 1842

The same year that Adams entered college he was apprenticed to William Hartigan, a leading surgeon in Dublin On Hartigan's death in 1813 Adams apprenticed himself to George Stewart, who at that time was Surgeon-General of the English army in Ireland In 1815 Adams was licensed by the Royal College of Surgeons in Ireland, and in 1818, at the age of twenty-seven, he was elected a member of that organization From then on he was very active in the practice of his profession

At an early date Adams was appointed surgeon to the Jervis Street Hospital in Dublin When Ephraim McDowell[1] died, Adams was selected to succeed him at Richmond Hospital in the same city The appointment was contested by John McDonnell and the claims of the two candidates were so evenly balanced that the board experienced great embarrassment as to whom they should select Richard Carmichael, who was surgeon to the hospital, solved the difficulty by resigning his surgical post to create a second vacancy, declaring that he was unwilling that the institution should be deprived of the services of either candidate, so highly did he estimate the merits of both men

At some time before his appointment to the Richmond Hospital, Adams, in conjunction with Kirby and Read, had founded the Peter Street School of Medicine He later broke his connection with the school But later, at Richmond Hospital, he founded another school, this time with Carmichael and McDowell This school was later known as the Carmichael School of Medicine and Surgery There Adams lectured for many years and, while thus occupied, wrote many admirable essays on

[1]Possibly a relative of the American Ephraim McDowell the ancestors of the American were from North Ireland See Schachner August *Ephraim McDowell Father of ovariotomy and founder of abdominal surgery with an appendix on Jane Todd Crawford* Philadelphia 1921 J B Lippincott Co xviii 331 pp

abnormal conditions of the joints These were published in Todd's "Cyclopaedia"[2] He also contributed several important articles on diseases of the heart These were published in the "Dublin Hospital Reports"

It was in 1826, when he was a surgeon to the Jervis Street Hospital, that Adams noticed the condition now known as "heart block" and frequently called "the Adams-Stokes syndrome" We are reproducing Adams' account, as well as that of Stokes According to Major,[3] heart block had been described by Marcus Gerbezius in 1719, Morgagni in "De Sedibus" (1761) and Thomas Spens in 1793. Adams' description, however, was the first complete account of this disease entity

In 1857 he published his classic account of rheumatic gout[4] In this publication he elaborated his views on chronic rheumatic arthritis, from which he himself suffered for many years previous to his death In 1861 Adams was appointed surgeon in ordinary to Her Majesty, Queen Victoria, and in the same year he was appointed Regius professor of surgery in Trinity College of Dublin

Adams was well versed in the writings of Continental surgeons and repeatedly referred to them in his lectures, writings, and even in his consultations He was much respected by his confrères, who once elected him president of the Dublin Pathological Society, and three times (1840, 1860 1861, 1867-1868) elected him president of the Royal College of Surgeons of Ireland

Adams died in January, 1875, presumably of cardiac disease He was buried in Mount Jerome Cemetery in Dublin

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[2]Todd R B *The cyclopaedia of anatomy and physiology*, London, 1835-1859, Longman [and others] 5 volumes in 6

[3]Major, R H *Classic Descriptions of Diseases*, Springfield, Ill, 1932 Charles C Thomas, pp 291-296

[4]Adams Robert *Treatise on rheumatic gout, or chronic rheumatic arthritis of all the joints*, London 1857, J Churchill 362 pp

CASES OF DISEASES OF THE HEART, ACCOMPANIED WITH PATHOLOGICAL OBSERVATIONS*

By

ROBERT ADAMS, A.B.

Member of the Royal College of Surgeons in Ireland, and one of the Surgeons to Jervis Street Infirmary, etc

The following case, in many particulars, and in its termination, resembled that above alluded to [the case published by J Cheyne, in 1818, A case of Apoplexy, in which the Fleshy Part of the Heart Was Converted into Fat]

An officer in the revenue, aged 68 years, of a full habit of body, had for a long time been incapable of any exertion, as he was subject to oppression of his breathing and continued cough In May, 1819, in conjunction with his ordinary medical attendant, Mr Duggan, I saw this gentleman he was just then recovering from the effects of an apoplectic attack, which had suddenly seized him three days before He was well enough to be about his house, and even to go out But he was oppressed by stupor, having a constant disposition to sleep, and still a very troublesome cough What most attracted my attention was, the irregularity of his breathing, and remarkable slowness of the pulse, which generally ranged at the rate of 30 in a minute Mr Duggan informed me that he had been in almost continual attendance on this gentleman for the last seven years, and that during that period he had seen him, he is quite certain, in not less than twenty apoplectic attacks Before each of them he was observed, for a day or two, heavy and lethargic, with loss of memory He would then fall down in a state of complete insensibility, and was on several occasions hurt by the fall When they attacked him, his pulse would become even slower than usual, his breathing loudly stertorous He was bled without loss of time, and the most active purgative medicines were exhibited As a preventive measure a large issue was inserted in the neck, and a spare regimen was directed for him He recovered from these attacks without any paralysis Oedema of the feet and ancles came on early in December, his cough became more urgent, and his breathing more oppressed, his faculties too became weaker

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*Published in Dublin Hospital Reports 4 353-453 1827 We reprint from Medical Classics 3 696, 1939 —J A M 1940

November 4th, 1819, he was suddenly seized with an apoplectic attack, which in two hours carried him off, before the arrival of his medical attendant

DISSECTION

56 hours after death

The dura mater presented a natural appearance The arachnoid membrane was separated from the pia mater by a fluid of gelatinous appearance The substance of the brain was watery and of a yellowish white colour There was some water in the ventricles These cavities did not appear enlarged, but the foramen of communication between them was dilated The coats of the carotid and middle arteries of the dura mater were quite white and opaque from bony deposition, but were pervious

The right lung was sound The left was compressed, and adhered to the side of the thorax, about a pint of serum and quantities of soft fat, of a very deep yellow colour, filled up the space between the anterior mediastinum and the compressed lung, which was impervious to air, and must have been totally useless

The right auricle of the heart was much dilated The right ventricle externally presented no appearance whatever of muscular fibres, it seemed composed of fat through almost its whole substance, of the same deep yellow colour as that which occupied the place of the left lung The reticulated lining of the ventricle, which here and there allowed the fat to appear between its fibres, alone presented any appearance of muscular structure

The left ventricle was very thin, and its whole surface was covered with a layer of fat Beneath this, the muscular structure was not a line in thickness, it had degenerated from its natural state, was soft, and easily torn, and a section of it exhibited more the appearance of liver than of a heart The septum of the ventricles presented the same appearance In both ventricles, even in the lining fibres, yellow spots, where fat had occupied the place of muscular structure, were to be observed The whole organ was remarkably light, the valves were all sound, except those of the aorta, which were studded with specks of bone, but elsewhere were cartilaginous and elastic, from which they derived a disposition to remain closed, a fluid gently injected from the ventricle would pass them, still, when the heart was reversed and water poured from the ventricle upon them, their valves retained it, its weight was not sufficient to separate the edges of the thickened valves There was much fluid blood contained in the heart

The liver was natural, the vena porta was unusually distended The spleen was healthy in its structure, although enlarged, the other viscera presented nothing unusual

In both these cases, No 1 and No 2, apoplexy must be considered less a disease in itself than symptomatic of one, the organic seat of which

was in the heart, although during life there was much analogy in their symptoms, the examination of the bodies after death disclosed a state of the heart altogether different, in one the ventricle was found nearly an inch in thickness, while in the other, fat had so accumulated at the expense of the muscular structure, that it was scarcely a line in depth The explanation of the fact how causes so different could have produced effects nearly similar, will, I imagine, be found in the reflection, that anything occasioning an undue distention of the vessels of the brain, may be followed by apoplexy This over distention may arise from the impulse a tergo being preternaturally strong, or on the contrary, it may be the result of some obstruction in front, as that arising from a contracted arterial opening, or some state of the ventricle incapacitating it from emptying itself with sufficient quickness to relieve the brain Indeed, upon considering the latter condition of things, where the heart is slow in transmitting the blood it receives, we find, I imagine, even in this a means of accounting for the lethargy, loss of memory, and vertigo, which attends these cases For the venous blood, which under such circumstances, is supposed to accumulate in the brain, is evidently ill-suited to the functions of this organ Although the quality of the blood may thus be supposed to have some influence in producing these bad consequences, yet it is probable that the principal causes determining an apoplectic attack where the heart is either actively enlarged, or in a state of atrophy, are mechanical and referable to circumstances in the heart, directly or indirectly producing a state of congestion of the vascular system of the brain

1831

JAMES HOPE

DESCRIPTIONS OF CARDIAC ASTHMA, STENOSIS OF THE PULMONARY VALVES, AND CARDIAC NEUROSIS

JAMES HOPE

Engraving from Memoir of the late James Hope, M D , by Mrs James Hope, published in 1848

(Courtesy Charles C Thomas)

JAMES HOPE

(1801-1841)

"[He] attained great eminence, and large practice, at an age when most physicians are only beginning to be heard of. His success was not owing to the patronage of any great man, nor to any of those fortunate accidents which have occasionally brought physicians suddenly into notice. He was indebted simply to his own talents, his active humanity, the weight of moral character, and the force of industry, for his rapid elevation."

—Dr. Klein Grant, in Preface to
Memoir of the late James Hope

JAMES HOPE was born in Stockport in the county of Cheshire, England, on February 23, 1801. His father was a successful merchant and manufacturer and desired his son, James, to become a merchant. The young man was educated at the Macclesfield Grammar School, and at the age of eighteen, he decided to become a lawyer. In the year 1819 occurred the Manchester riots, culminating in the so-called Battle of Peterloo on August 16, which so enraged the poet, Percy Bysshe Shelley, that in his "Mask of Anarchy, Written on the Occasion of the Murder at Manchester," he excoriated England's foreign secretary of the time, Robert Stewart Castlereagh, in the lines:

> "I met Murder on the way—
> He had a mask like Castlereagh."

In 1822, when Viscount Castlereagh cut his throat, Lord Byron wrote:

> "So He has cut his throat at last!—He! Who?
> The man who cut his country's long ago."

Young Hope enlisted in, and spent about a year with, the Yeomanry Lancers, a body of men raised to cope with the disturbance. When he subsequently returned home, his father suggested that he become a physician. Hope did not like this idea, but decided to give medicine a trial provided, after he became a physician, he should be allowed to practice in London.

In 1820, after a period spent at Oxford, Hope began the study of medicine at Edinburgh. The next year he was asked by Dr. James Bardesley of Manchester to join the Royal Medical Society of Edinburgh. At a meeting of this society he presented a paper on the heart, and it was so well received that he decided to write a work on diseases of the heart.

In 1824, Hope was elected house physician to the Edinburgh Royal Infirmary, and in 1825 became house surgeon to this institution. Later, that same year, he received the degree of Doctor of Medicine from the University of Edinburgh. He chose as his dissertation subject "Aneurism of the Aorta." In his thesis (1825) he proved that it was possible to diagnose aneurysms of the aorta during the life of the patient which, according to Laennec, was not possible.

Hope, feeling that it would better qualify him for the practice of medicine, went to London to study surgery at St. Bartholomew's Hospital, in 1826. In the spring of

that year he passed his examination in surgery before the College of Surgeons in London He spent the next year in Paris, where he visited many of the hospitals He settled for some time at La Charité, where he worked as clinical clerk under Dr Auguste-François Chomel

In July, 1827, Hope left France to travel and study in Switzerland and in Italy He returned home in June, 1828, and in December of that year began the practice of medicine in London In 1829 he established a private dispensary, which was maintained for three years until his appointment as physician to the Marylebone Infirmary in 1831 In order to gain more experience, he registered as a student at St George's Hospital where he later was to become physician In March of 1831 his marriage to Anne Fulton took place Mrs Hope was later to write a "Memoir" of her distinguished husband

During the early years of his practice, Hope, in order to determine the cause of the heart sounds, experimented on donkeys He succeeded in examining the hearts of stunned asses, in which respiration had been artificially maintained after the pericardium was opened, and proved that the second heart sound was dependent on the abrupt closure of the aortic and pulmonic valves

Evidence of his continued interest in heart disease came with the publication, in 1831, of his "Treatise on the Diseases of the Heart and Great Vessels" Therein is contained the notation of the "jerking" pulse of aortic insufficiency, which Cowper had described in 1705 and Vieussens had described in 1715 and which Corrigan, as we have shown, was to describe in more detail in 1832 The third edition of this work was published in England in 1839 and it first appeared in the United States in 1842

Since the American edition of his work is considered to be the best one, we have chosen to reprint from it the following important descriptions (1) description of cardiac asthma (according to Flaxman, he was the first to introduce this term), (2) stenosis of the pulmonary valves, and (3) cardiac neurosis

Another important work by Hope was his "Principles and Illustrations on Morbid Anatomy," which was published in parts, and was completed in 1834

Many of Hope's brothers and sisters were victims of tuberculosis, and although he showed no evidence of this disease in February, 1836, when he was examined as a candidate for life insurance, he was destined to die of it five years later In May, 1836, he was afflicted by a slight cough accompanied by a pain in the thorax The following year he suffered an attack of influenza, and from that time on the cough persisted

In August, 1838, Hope went to Scotland for a month's vacation His health appeared to improve following this rest, but when he returned to work he suffered a relapse He was advised to give up his work and to travel, but because he desired to be appointed physician to St George's Hospital, he refused to abide by this advice

On July 5, 1839, he received the appointment, which had been a long-cherished wish His health, although it seemed at times to be better, gradually declined, and he died on May 13, 1841, at the age of forty

A TREATISE ON THE DISEASES OF THE HEART AND GREAT VESSELS*

By

J. HOPE, M.D., F.R.S.

Of St George's Hospital, formerly Senior Physician to the St Marylebone Infirmary, Extraordinary Member, and formerly President, of the Royal Medical Society of Edinburgh, etc

CARDIAC ASTHMA†

AMONGST the diseases of the heart may be justly reckoned one of the forms of the malady termed in common language *asthma* This has been too much regarded as independent of disease of the heart Long treatises have even been written upon it without ever mentioning disease of this organ as one of its causes It is, therefore, necessary to notice the subject formally in this place, not only for the purpose of showing the magnitude of the error, but of making the reader acquainted with all the habitudes and aspects of a complaint, which is perhaps the most distressing in the whole catalogue of human maladies

It is established by the concurrent testimony of all moderns conversant with diseases of the heart, that these diseases, no less than those of the lungs, may constitute the organic causes of asthma

A theoretical consideration of the subject leads, in my opinion, to the same conclusion, for, on tracing asthma back to its source, we shall find that, whatever be its proximate cause in different cases, it is connected, in all, with the same ultimate circumstance, namely, inadequate oxygenization of the blood, and the resulting want of breath, which, through the "incident excito-motory" branches of the pneumogastric, excites the "reflex" action of the "true spinal" nerves on the muscles of respiration For instance, inadequate oxygenization of the blood results in all ordinary cases from one or more of three proximate causes viz

A Insufficient admission of air into the bronchial tubes and air-vesicles

B Insufficient exposure of the blood to the air admitted, in consequence of a less pervious state of the mucous membrane than natural

C Insufficient admission of blood into the lungs

It will be found that, to one or more of these causes, all the varieties of dyspnoea and asthma are referable

*First English edition 1831 We reprint from the first American edition Philadelphia Haswell and Johnson 1842 pp 376-384 —F A W 1940

†From Part III Chap IX Section 5

4. *Diseases of the Heart*.—Sometimes, from this cause, blood exists in the lungs in excess; as is the case when the right ventricle is hypertrophous, or the left side of the heart obstructed, or, still more, when these two affections co-exist; also when the circulation is merely accelerated, as by palpitation, running, or by slighter efforts in corpulent persons. Now, under all these circumstances, there is inadequate oxygenization of the blood, or, in other words, there is an excess of venous blood in the lungs; first, because the quantity of blood admitted exceeds its due proportion to the air in the organ; secondly, because the engorgement of the mucous membrane on which the blood ramifies, constricts the bronchial passages, and prevents the free ingress of air, as proved by the feebleness of the respiratory murmur. Hence, want of breath is a necessary consequence of an excess of blood in the lungs.

Sometimes blood does not enter the lungs in sufficient quantity, constituting the third cause of inadequate oxygenization; and this may arise from the weakness of the right ventricle, from an obstruction in its mouth, or from increased resistance on the part of the lungs; as, for instance, during sleep, when the respirative function is less active. Hence results the stimulus of want of breath, and dyspnoea. Cases exemplifying this will shortly be adduced: meanwhile it may be illustrated by a simple physiological experiment, viz. by making and sustaining a full *expiration*. This is attended, not only with a deficiency of air, but also with a deficient influx of blood into the lungs, as is proved by the lividity of the face which ensues, by the elevation of the fontanel in infants, by the rise of blood in a tube inserted into the jugular vein; and lastly by experiment; for I have demonstrated above, that, on suspending artificial respiration in a rabbit, the heart *instantly* became gorged, of a black colour, and distended to nearly double its natural size—a phenomenon which renders it sufficiently manifest that, when the lungs are exhausted of air, the blood does not freely enter them. Now, the sensation of want of breath experienced on making a full expiration is familiar to every one, and it becomes intolerable if the expiration be long sustained.

5. *Spasmodic constriction of the bronchial tubes* is presumed to exist, first, because, according to the researches of Reisseissen and others, the bronchial tubes are provided with muscular fibres, and all muscles are liable to spasm: secondly, because asthma is occasionally found to occur without any organic cause (so far at least, as our senses enable us to judge) sufficient to account for it: thirdly, because every form of organic disease above described, both of the lungs and the heart, may exist without causing dyspnoea of such intensity and of such a character as to constitute *asthma* properly so called. Thus, many have intense chronic bronchitis and profuse expectoration without any asthmatic dyspnoea; and I have known a patient with a contraction of the mitral orifice to the size of a small pea, and likewise with dilatation and softening of the heart and profuse expectoration,

pass through a period of ten years to her grave without ever experiencing a paroxysm of asthma, though a few steps across the room were sufficient to excite dyspnoea (Mrs —l—n)

Hence, I apprehend that whatever be the organic cause of asthma, it requires for its production the superaddition of a state of the nervous system leading to spasmodic constriction of the bronchial tubes Why some should exhibit this state and others not, is one of the arcana of the nervous system, but observation has shown that the state is constitutional and often hereditary

Admitting that the spasmodic constriction of the bronchial tubes does take place, it is obvious that it will more or less close these tubes against the ingress of air, and this closure, again, by preventing the free expansion of the lungs, will impede the influx of blood Whence there is a double cause for the inadequate oxygenization of the blood, and consequently, for the production and maintenance of the asthmatic paroxysm

From all that has been said, we are now led to the resulting inquiry—what is the essential difference between asthma from disease of the heart and that from disease of the lungs Putting aside that variety of asthma which, as not being attended with any *visible* organic derangement, (though it is, notwithstanding, highly probable that one exists,) may be regarded as mainly, if not wholly spasmodic, there does not appear to be any essential difference between the remaining varieties Their organic causes are diversified, but they all ultimately produce the same effect, and it is the effect which constitutes the essence of the disease This effect is inadequate oxygenization of the blood, which causes "excitant" want of breath, and this, when the case is really asthmatic, i e more than what may be called mere dyspnoea, occasions spasmodic constriction of the bronchial tubes, and its consequence, the asthmatic paroxysm

We now proceed to the more particular consideration of asthma from disease of the heart

This variety comprises, according to my observation, by far the greater proportion of the most severe and fatal cases of the disease Some are of the opinion that in other varieties the patient experiences an equal degree of suffering during the continuance of the paroxysm I cannot say that this is consistent with my own observation Though the same words may suit for the delineation of an attack of each variety, my feeling and conviction is, that I have never seen the patient suffer such intense and suffocative agony as in the variety from organic disease of the heart

Until the discovery of auscultation had in some degree dissipated the deep obscurity of the affections of this organ the fact that they were a cause of asthma was scarcely known and, even at the present day, there are few errors more common than that of attributing asthma to other causes, when it originates solely in the heart For instance a theory of this description which has for the last half century been more widely disseminated than

perhaps any other, consists in ascribing asthma to a spasmodic or convulsive contraction of the external muscles of respiration, much dependent on habit.

Now, the action of these muscles, so far from being morbid or dependent on habit, is a natural instinctive and salutary effort to prevent suffocation, the stimulus to which consists in an exaggeration of that which excites the muscles in ordinary respiration—namely, as above explained, the want of breath, resulting from inadequate oxygenization of the blood. Nothing is more common, for instance, than to see a patient with diseased heart, while sleeping tranquilly, start up and begin to respire with violence. Here it is obvious that the necessity for violent respiration preceded the act, and the necessity depends on impeded transmission of blood through the heart and lungs, for starting is invariably accompanied by palpitation, and preceded by frightful dreams, or some sensation of praecordial distress, indicating an obstructed circulation. I have frequently examined the heart and lungs by auscultation immediately before the supervention of a paroxysm of dyspnoea, and have always found that the heart began either to palpitate, or to act in that irregular, confused, and, as it were, struggling manner, which denotes its engorgement. I was therefore enabled to tell the patient that difficulty of breathing was coming on, to which with some astonishment, he would reply in the affirmative, being himself forewarned of the approaching accession by a feeling of anxiety and straitness in the praecordia. The fact is so universally true, that any one may satisfy himself of it by entering an hospital and gently placing a patient with orthopnoea from disease of the heart, in a rather uneasy position, when the series of phenomena described will become manifest.

Dr. Burrows communicated to me the particulars of a case, recently under his observation, in which the respiration was alternately violent and tranquil under the following circumstances. The patient dozed for a few minutes at a time, during which his complexion became livid, and his pulse more and more feeble, oppressed and irregular. He then started up, and, after a few violent wheezing respirations, relapsed into the same calm doze. In this case the mitral orifice was contracted to the size of a pea. Now, there can be little doubt that as, during sleep, the stimulus of want of breath is less felt, and the muscles of respiration are, consequently, less excited by it,—in simple language, as the respiration is more feeble during sleep, the lungs were not, in the present case, kept sufficiently expanded to admit of an adequate circulation through them; whence ensued engorgement of the heart and venous system of the body, with insufficient arterialization of blood in the lungs, and the necessity for breathing resulting from it, which series of phenomena was relieved by the succeeding violent respirations. I have frequently observed this series of phenomena in a greater or less degree; occasionally even in coma. In another case, violent gasping and wheezing respiration, lasting from a few seconds to two or three minutes,

occurred at intervals of four or five minutes, during which the patient dozed, even though sitting erect on a stool and undergoing a stethoscopic examination, and this series of actions continued so long as the patient remained disposed to sleep in that situation In another case of great dilatation and softening, the precise symptoms described by Dr Burrows occurred for the last week of the patient's life, whether he was awake or asleep, except when calmer sleep was procured by mild opiates In a third case, a lady had, for several years, observed her husband's respiration while he was in the horizontal position, but not in the raised position, to be as follows —after every four or five respirations calmly performed, succeeded a pause of a few seconds, then he started with a "convulsive motion of all his limbs, and a heaving of the shoulders" She had watched this continue for hours together, but he was unconscious of it, and generally slept soundly without frightful dreams His disease was slight hypertrophy and disease of the aorta

In all these cases, it is manifest that the action of the muscles of respiration was consecutive to the obstruction of the circulation, and that it was not dependent on any spasm of those muscles, but simply on the necessity for breathing, which instinctively excited them to a salutary preservative effort

Asthma from disease of the heart often imitates the characters of the other varieties, and this perhaps for a very simple reason, that the lungs are in much the same state as in those varieties Thus, it is *humid* or *humoral*, when there is permanent engorgement of the lungs, causing copious sero-mucous effusion into the air-passages, as in cases of contraction of the mitral valve It is *dry*, when the engorgement is only temporary, as in cases of pure hypertrophy It is *continued*, when there is a permanent obstruction to the circulation, and any of the varieties may be *convulsive*, when the heart has sufficient power to palpitate violently The worst cases of convulsive asthma from disease of the heart are those of hypertrophy with dilatation and a valvular or aortic obstruction

We shall now examine the state of a patient labouring under severe asthma from disease of the heart, and then take a more strictly medical view of the nature and progress of the asthmatic paroxysm

The respiration, always short, becomes hurried and laborious on the slightest exertion or mental emotion The effort of ascending a staircase is peculiarly distressing The patient stops abruptly, grasps at the first object that presents itself, and fixing the upper extremities in order to afford a fulcrum for the muscles of respiration, gasps with an aspect of extreme distress

Incapable of lying down, he is seen for weeks, and even for months together either reclining in the semi-erect posture supported by pillows, or sitting with the trunk bent forwards and the elbows or fore-arms resting on the drawn-up knees The latter position he assumes when attacked by

a paroxysm of dyspnoea—sometimes, however, extending the arms against the bed on either side, to afford a firmer fulcrum for the muscles of respiration. With eyes widely expanded and starting, eye-brows raised, nostrils dilated, a ghastly and haggard countenance, and the head thrown back at every inspiration, he casts round a hurried, distracted look of horror, of anguish, and of supplication: now imploring, in plaintive moans, or quick, broken accents, and half-stifled voice, the assistance already often lavished in vain, now upbraiding the impotency of medicine, and now, in an agony of despair, drooping his head on his chest, and muttering a fervent invocation for death to put a period to his sufferings. For a few hours—perhaps only for a few minutes—he tastes an interval of delicious respite, which cheers him with the hope that the worst is over, and that his recovery is at hand. Soon that hope vanishes. From a slumber fraught with the horrors of a hideous dream, he starts up with a wild exclamation that "it is returning." At length, after reiterated recurrences of the same attacks, the muscles of respiration, subdued by efforts of which the instinct of self-preservation alone renders them capable, participate in the general exhaustion, and refuse to perform their function. The patient gasps, sinks, and expires.

Such are the sufferings, in their worst form, of an asthmatic from disease of the heart. We have now to take a more strictly medical view of the nature and progress of the asthmatic paroxysm.

If about to be severe, it is generally preceded by certain premonitory symptoms, which, though not so marked as in ordinary asthma, are much of the same nature—probably because derangement of the circulation and imperfect oxygenization of the blood are present in both. In cardiac asthma, however, many of the nervous symptoms which characterise the ordinary varieties are often deficient. One of the most common and efficient exciting causes of cardiac, as of all other asthmas, is derangement of the stomach, the irritation of which extends to the heart, and stimulates it to inordinate action. The irritation, according to the theory of Sir Charles Bell, or the lately revived excito-motory views of Prochasca, is propagated through the medium of the par vagum, by which nerves the stomach and heart are closely associated. Accordingly, after a feeling of acidity, flatulence, or a load on the stomach from undigested food, often accompanied with abdominal distention, the patient experiences pain, weight, and constriction in the forehead and over the eyes, accompanied (if the case be one of hypertrophy of the left ventricle) with throbbing of the temples and the sound of rushing waters. He feels a sensation, scarcely to be defined, of oppression, and tightness and anxiety about the praecordia, frequently with slight palpitation. Sometimes the patient is drowsy, listless, restless, irritable, and impatient, not only of society, but of the attentions of friends: these symptoms, however, are, in general, more prevalent in ordinary asthma. The signs described afford the experienced asthmatic well-known assurance of the approaching attack.

They gradually become worse and worse, especially after a meal, and eventually burst into a paroxysm The time of the accession is less regular than in ordinary asthma, being more dependent on the state of the heart, which is liable to accidental excitement at any moment, from a variety of causes The fit, however, as in ordinary asthma, is, on the whole, more apt to supervene during the evening or early part of the night, and this, as appears to me, for two reasons 1st The recumbent position is unfavourable to respiration, the diaphragm being pressed upwards by the abdominal viscera, and the expansion of the chest being opposed by its own weight 2d During sleep, respiration is not assisted by the will, which, during the wakeful state, from the sensation of want of breath being more acutely felt, is ever ready to maintain the body in the position most favourable to breathing From the co-operation of these two causes, therefore, the circulation becomes so far embarrassed before the patient is aroused to a sense of his condition, that it can only be relieved by those violent efforts which constitute the asthmatic paroxysm He accordingly awakes, generally with a start, in a fit of dyspnoea, accompanied either with violent palpitation, or a distressing sense of anxiety in the praecordia and great constriction of the chest, as if it were tightly bound He is compelled to assume a more erect posture, and intensely desires fresh, cool air, the respiration is wheezing, and performed with violent efforts of all the muscles of respiration, both ordinary and auxiliary The inspirations are high and accompanied with apparently little descent of the diaphragm, and the expirations are short and imperfect The surface is chilly, the extremities are cold, and the face is pale and sometimes livid

In cases in which the pulmonary congestion is only *temporary*, as in hypertrophy either simple or with dilatation, there is no cough beyond a few slight and ineffectual efforts, producing little or no expectoration, and in such cases the fit subsides as soon as the engorgement of the heart and great vessels is relieved, which nature generally effects in two or three hours or less, by determining the blood to the surface and creating diaphoresis In some instances, I have known this to be regularly accompanied with a copious secretion of pale urine and a purging alvine evacuation (case of May) In this case, the attacks recurred, according to the assertion of the patient, every night for several years

The pulse, however full, strong and bounding at first, may, during the worst of the paroxysm, become feeble and small, and the sound and impulse of the heart may be diminished, and this, in cases even of hypertrophy, for the organ, being gorged to excess, is incapable of adequately contracting on its contents

Such is the nature of an asthmatic fit when the pulmonary congestion is only temporary the case is different when it is *permanent*, as in valvular disease and in some extreme cases of dilatation For then, there is violent cough in suffocative paroxysms, accompanied, at first, with difficult and

scanty expectoration of viscid mucous, but ending gradually in a copious and free discharge of thin, transparent, frothy fluid, occasionally intermixed with blood. This evacuation, by disgorging the pulmonary capillaries, affords great relief to the cough and dyspnoea. As, however, the transudation of the matter to be expectorated into the air-passages, and its final elimination, are slow processes, paroxysms of this description are much more protracted than those of dry asthma from hypertrophy. They frequently last five or six hours, and I have known them to persist, with occasional remissions, for two, three, or more days. During the attack, the pulse is quick, small, and weak, often irregular and intermittent. The slowness which the latter characters sometimes appear to give it, has led some authors to suppose that the circulation through the heart is little disturbed in asthma. This is in some degree true in reference to other varieties of asthma, but it is always incorrect in reference to that from disease of the heart.

As the paroxysm subsides, the anxiety and constriction decrease, the respiration becomes less frequent, high, and laborious, and the pulse becomes slower, fuller, and more regular. But some degree of wheezing and tightness of the chest remain, and the paroxysm is very apt to return for two or three nights successively, and sometimes for a much longer period, until the lungs are freely unloaded by copious expectoration. It may, indeed, continue to recur at brief intervals for an indefinite period, or the patient may never be wholly exempt from some degree of asthmatic dyspnoea.

A severe asthmatic attack from disease of the heart is in general far more injurious in its consequence than one from an affection of the lungs.

PART III, CHAPTER IX

SECTION IV

*SIGNS OF DISEASE OF THE PULMONIC VALVES**

The signs of contraction of the pulmonic valves are the same as those of the aortic, with this difference, that, from the vessel being nearer the surface the murmur with the first sound seems *closer* to the ear, and is on a higher key, ranging from the sound of a whispered *r* towards that of *s*. I have, however, known it fall below *r* when the circulation was feeble and slow, and the obstruction slight. It may be known that the murmur is not seated in the aorta, by its being inaudible, or comparatively feeble, two inches up that vessel, whereas, at a corresponding height up the pulmonary artery, it is distinct; also, by its being louder down the tract of the right ventricle than down that of the left (Bowden). It may be known that the murmur does not proceed from regurgitation through the auricular valves,

*Ibid pp 368-369

by its being distinct along the course of the pulmonary artery, where auricular murmurs are either wholly inaudible, or very feeble and remote

When a murmur in the pulmonary artery is considerably louder between the second, and third left ribs, close to the sternum, than opposite to the valves, and is there attended with impulse and purring tremor, dilatation of the pulmonary artery may be suspected (see *Dilatation of Pulmonary Artery*) In one instance I have known a murmur to be produced by complete ossification of the pulmonary artery penetrating deeply into the lungs (case of Lady R)

When there is regurgitation through the pulmonic valves, a murmur accompanies the second sound Its nature and diagnosis are the same, (the necessary inversions being made,) as in the case of aortic regurgitation, except that the pulse is not jerking (case of Rogers A tremor attended)

I presume that purring tremor with the first sound may be occasioned by contraction of the pulmonic orifice, though I have not met with an instance verified after death but I have met with three in which the tremor attended dilatation of the pulmonary artery (Weatherly, Bowden, and Miss L P—r) A purring tremor occasioned by the pulmonic valves would be more readily felt than one occasioned by the aortic valves, because it would probably be transmitted as far as the space between the second and third ribs, (where it is out of the cover of the sternum,) provided the patient lay in the horizontal position, and inclined to the left side

Disease of the pulmonic valves is so rare, that it ought never to be suspected unless the signs described are perfectly well marked, or unless there be patescence of the foramen ovale, or some other communication between the two sides of the heart,—states which experience has proved to be generally accompanied with contraction of the orifice in question

PART IV, CHAPTER II

PALPITATION FROM INORGANIC CAUSES, USUALLY CALLED NERVOUS, AND IMITATING DISEASE OF THE HEART*

There are few affections which excite more alarm and anxiety in the mind of the patient than this He fancies himself doomed to become a martyr to organic disease of the heart, of the horrors of which he has an exaggerated idea, and it is the more difficult to divest him of this impression, because the nervous state which gives rise to his complaint, imparts a fanciful, gloomy and desponding tone to his imagination Members of the medical profession are more apt than others to give way to these feelings, partly from their apprehensions being more keen and partly from an impression too widely prevalent that there is difficulty in distinguishing inorganic from organic palpitation and, consequently, that they must remain in a

*Ibid pp 468-470

state of anxious uncertainty It may be said, for the consolation of such, that the diagnosis presents no difficulty to one who, to general signs, adds a knowledge of these afforded by auscultation and percussion I repeat this opinion with increased confidence in the present edition, not only on the grounds of additional experience, but because the signs both of organic and inorganic disease will now be found much more precise and simple, in consequence of the new lights thrown on particular valvular diagnosis and on inorganic murmurs

Inorganic palpitation presents certain varieties, which it is of the greatest practical importance to distinguish, as the treatment is different, and even opposite It may be premised that, in all the varieties, the palpitation will, caeteris paribus, be greater in proportion as the patient is constitutionally of a more nervous, irritable temperament

1 Palpitation dependent on dyspepsia, hypochondriasis, hysteria, latent gout, mental perturbations either of the exciting or depressing kind, excessive study with deficient sleep, and venereal excesses, constitutes the first variety, and forms a large class When from these causes, it presents various degrees and characters The slightest degree of it I should describe, from having occasionally experienced it, to be a tumbling or rolling motion of the heart, with a momentary feeling of tightness and oppression It is referable to an intermission of the heart's action In a further degree, as Abercrombie has well described, there is a series of quick, weak, fluttering, irregular beats, with slight anxiety, acceleration of the respiration, and a quivering sensation in the epigastrium this may last from a few minutes to half an hour or an hour, and occur only at distant and irregular intervals, or repeatedly during the day, especially when the patient is startled The next degree amounts to a perfect fit of palpitation, consisting in increased impulse, sound and frequency of the beats, sometimes with irregularity, and generally with more or less anxiety, dyspnoea, and even orthopnoea The attack may be only occasional, or may occur several times a day, or may even last with little intermission for several days together

The palpitation in question may be distinguished from that of disease of the heart, by the palpitation occurring only occasionally by its not being excited, but, on the contrary, relieved by corporeal exercise of such a nature as would certainly disturb the action of a diseased heart by its disposition to supervene while the patient is at rest, especially at the commencement of the night, when he lies wakeful in bed, by a fluttering in the epigastrium, by the general prevalence of nervous symptoms, by the affection being aggravated when the nervous symptoms undergo an exacerbation, by the pulse and the action of the heart being natural during the intervals between the attacks, and by the absence of valvular and aortic murmurs, and of undue impulse, "the shock, even when it at first appears strong, having little real impulsive force, for it does not sensibly elevate the head of the observer" (Laennec)

To this category some would add, an increase of the palpitation after meals, or when the stomach is deranged, and amelioration produced by dyspeptic remedies, but, as the stomach produces the same effects when there is disease of the heart, these signs are not pathognomic of nervous palpitation. To this point I would particularly direct the attention of practitioners, because many, in forming their diagnosis of the affections in question, regard the dyspeptic signs as paramount in value to all others, and are apt to refer to the stomach the palpitation which really belongs to organic disease of the heart.

Though the present variety of palpitation is often attended with various familiar nervous affections of the head, as pain or sensations of heat or of cold confined to particular parts and coming and going suddenly, temporary vertigo, tinnitus, and confusion of the sight, not increased by lying or stooping, it is not, when purely nervous and the patient not plethoric, accompanied with genuine signs of cerebral determination or congestion. there is no universal, throbbing headache with weight and tension, increased by stooping or the recumbent position. no stunning sounds and pains in the head on suddenly lying down or rising up. no permanent somnolency, apoplectic stupor, or regular apoplectic fits, as in hypertrophy, etc.

When it has been ascertained that the palpitation in question is independent of organic disease, the treatment presents no unusual difficulty, and is to be adapted to the nature of the exciting causes specified at the head of this division. It would be foreign to the subject of this work to dwell upon the particular remedial measures.

1832

SIR DOMINIC JOHN CORRIGAN

DESCRIPTION OF THE PULSE IN AORTIC INSUFFICIENCY (CORRIGAN PULSE)

SIR DOMINIC JOHN CORRIGAN

(Courtesy Medical Classics)

SIR DOMINIC JOHN CORRIGAN

(1802 1880)

"We know no difference of race, or creed,
or colour, for every man is our neighbour"

—Dominic Corrigan, quoted by Williamson

DOMINIC JOHN CORRIGAN was born in Dublin on December 1, 1802 His father was a successful farmer who also sold agricultural implements Young Corrigan was educated at the lay College of Saint Patrick at Maynooth There he studied the classical languages, French, and natural science Corrigan distinguished himself in his studies at Maynooth and was frequently called upon to assist the professor of natural philosophy

Following his schooling at Maynooth, Corrigan was apprenticed to Dr O'Kelly, physician to the same college O'Kelly was impressed with Corrigan's ability and advised his father to send the young apprentice to study medicine at Edinburgh University

O'Kelly's advice was accepted and Corrigan completed his medical studies at Edinburgh He was graduated with the degree of Doctor of Medicine in 1825 It is interesting to note that he was a classmate of William Stokes, who was also destined to make significant contributions to the study of the diseased heart

Soon after graduation, Corrigan settled in Dublin where he continued to study as well as to practice medicine In 1830 he became physician to the Jervis Street Hospital While at Jervis Street Hospital, Corrigan wrote the two papers that were to make his name famous, "On Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves" (1832), and "On Cirrhosis of the Lung" (1838) In 1831 he was appointed physician to the college at Maynooth Corrigan was active in the treatment of cholera, that disease having appeared in Dublin in 1832 Elsewhere in the present volume we have related the experience of Stokes, who reported the first case of the epidemic

Corrigan was an excellent teacher He became professor of medicine first in the Digges Street School, next in the Peter Street School, and later in the Richmond Hospital (or Carmichael School) An important article by Corrigan, "An Aneurism of the Aorta" was published in the "Lancet" for February 7, 1829 This paper, his first published work, emphasized the value of the stethoscope in the diagnosis of cardiac conditions His article on aneurysms was soon followed by his "Inquiry into the Causes of 'Bruit de Soufflet' and 'Frémissement Cataire'" published in the "Lancet" for April 4 and April 11, 1829 Therein Corrigan showed that Laennec's conception of "bruit de soufflet" was erroneous Laennec believed that these sounds were the result of spasm, but Corrigan explained them on a purely physical basis Therein, too, is the first suggestion of what Chauveau and Savart long afterward described as "fluid veins," or eddies in the vessels

The immortality of Corrigan, in cardiology, rests of course on his famous paper on aortic insufficiency This paper it is our privilege to reprint It was published in 1832 when Corrigan was thirty years of age When this article was written, Corrigan honestly believed that it was the first published work on the subject James Hope took violent exception to the assumption and claimed the discovery of aortic insufficiency as his own, made in 1825 Hope claimed that he had taught this subject at St Bartholomew's Hospital in 1826 and at La Charité, Paris, in 1827[1] Hope[2] in 1831 had described the "jerking" pulse of aortic insufficiency More noteworthy cases of this condition had been observed earlier than Hope's description, however William Cowper,[3] in 1705, and Raymond de Vieussens, in 1715, had described the clinical aspects of aortic insufficiency, but they did not attempt to discover the pathologic cause Samuel Wilks[4] suggested that the most important claim was that of Thomas Hodgkin who, in 1827 and in 1829, had read two papers before the Hunterian Society in which he showed knowledge of the chief signs of aortic insufficiency However, none of the accounts mentioned equals that of Corrigan for completeness and masterful description

Another important paper of Corrigan's, published in 1837, in the "Dublin Journal of the Medical Sciences," was "On Aortitis as one of the Causes of Angina Pectoris" It is of interest to note that Rolleston said that Sir Clifford Allbutt, who advanced the same explanation in 1894, did not find out until 1908 that Corrigan had written about it seventy-one years previously

In addition to "Corrigan's pulse" or as the great French clinician, Armand Trousseau, named it, "maladie de Corrigan," another condition of pathologic importance was named after him This was long known as "Corrigan's cirrhosis," today better known as "fibroid disease of the lung" In his article on cirrhosis of the lung, published in the "Dublin Journal of Medical Science" for May, 1838, Corrigan demonstrated the difference between cirrhosis and tubercular phthisis

That same year (1838) Corrigan became one of the founders of the Dublin Pathological Society He later served as president of the organization The publication of these last two papers greatly increased Corrigan's reputation as a clinician, and in 1840 he was elected physician to the Whitworth Medical and the Hardwicke Fever Hospitals In 1843 he received his diploma as a member of the Royal College of Surgeons (London) On the invitation of the examiners he gave an oral dissertation on the patency of the aortic valves

In 1849 the honorary degree of Doctor of Medicine was conferred on him by the University of Dublin With the founding of Queen's University in 1850, Corrigan was made a member of the University Senate In 1859 he was its representative in the medical council, and in 1871 he was elected vice chancellor of the University

Corrigan held the office of president of the King's and Queen's College of Physicians for five consecutive years A colossal statue in white marble of him was made by Foley and it stands next to statues of his great contemporaries, Graves, Marsh and Stokes

In 1866 Corrigan was created a baronet, not only because of his high attainments in medicine, but also because of his services to the government In 1870 he was appointed physician-in ordinary in Ireland to Queen Victoria That same year he was made one of the commissioners of national education in Ireland

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[1] Dock, George Dominic John Corrigan Ann M Hist n s 6 381-395 1934

[2] Hope, James *A Treatise on the Diseases of the Heart and Great Vessels*, London 1831 Kidd, 612 pp

[3] Cowper's contribution is reprinted on pp 109-114

[4] Wilks Samuel Notes on the History of Valvular Disease of the Heart, Guy's Hosp Rep (s 3) 16 209-216 1871

At the age of sixty-eight, Sir Dominic was elected to represent Dublin in the British House of Commons He held his seat until the dissolution of Parliament in 1874 Corrigan took a great interest in zoology, was a member and later president of the Royal Zoological Society of Ireland, the Royal Irish Academy, the Academy of Medicine of Paris, and of the Harveian Society of London In 1875 he was elected first president of the Pharmaceutical Society of Ireland

Corrigan in his later years suffered from attacks of gout In 1878 he had a slight paralytic stroke and in 1880 he died at the age of seventy-eight following a massive cerebral vascular accident with right hemiplegia

ON PERMANENT PATENCY OF THE MOUTH OF THE AORTA, OR INADEQUACY OF THE AORTIC VALVES*

By

D J CORRIGAN, M D

One of the physicians to the Charitable Infirmary, Jervis Street, Dublin, Lecturer on the Theory and Practice of Medicine, Consulting Physician to St Patrick's College, Maynooth

THE disease to which the above name is given has not, so far as I am aware, been described in any of the works on diseases of the heart The object of the present paper is to supply that deficiency The disease is not uncommon It forms a considerable proportion of cases of deranged action of the heart, and it deserves attention from its peculiar signs, its progress and its treatment The pathological essence of the disease consists in inefficiency of the valvular apparatus at the mouth of the aorta, in consequence of which the blood sent into the aorta regurgitates into the ventricle This regurgitation, and the signs by which it is denoted, are not necessarily connected with one particular change of structure in the valvular apparatus, and hence the name *Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves*, has been chosen as simply expressing such a state of the parts as permits the regurgitation to occur

I have been in the habit for some years of describing this disease under the name of *Inadequacy of the Aortic Valves*, but as Dr Elliotson, in his elegantly written work on Diseases of the Heart, has given to a somewhat analogous morbid state of the auriculo-ventricular opening, a better name, *Permanent Patency,* I have, for that reason, and for the sake of uniformity, adopted the term, and I shall continue to use it as synonymous with my own term, *Inadequacy of the Aortic Valves*

The morbid affections of the valves and aorta permitting this regurgitation are the following

1*st*—The valves may be absorbed in patches, and thus become reticulated and present holes, through which the blood flows back into the ventricle—*Vid* Plate I, Fig 1

2*d*—One or more of the valves may be ruptured, the ruptured valves, when pressed, flapping back into the ventricle instead of catching and supporting the column of blood in the aorta, the blood then regurgitating through the space left by the broken valves—*Vid* Plate I, Fig 2

*Originally published in the Edinburgh Med and Surg J 37 225-245 1832 We reprint from Medical Classics 1 703-727 1937—F A W 1940

Fig 1

Fig 2

Fig 3

Fig 1 This figure scarcely needs any explanation It shows the reticulated valves The letters *A*, *A*, *A*, *A*, point out the openings produced by absorption in the valves, through which the blood regurgitated These valves were very slightly thickened

Fig 2 *A*, points out the left hand valve, with an opening through it large enough to admit a goose quill, and ruptured from its connection with the aorta, so that it flapped back into the ventricle

B Bony depositions on the inner coat of the aorta

C The middle and right hand valves thickened, and contracted in their free edges, so that they could be separated only a very short distance from the sides of the aorta

Fig 3 *A*, *A* Openings in the valves, as in Fig 1, produced by absorption, one of the openings in the right hand valve large enough to permit the finger to pass through

B Middle valve, projecting downwards, curled back, and bound to the aorta by bony deposition, so that it was totally useless

C Bony deposition tying the edges of the middle and right hand valve together, and at the same time gluing them to the aorta

3*d*.—The valves may be tightened or curled in against the sides of the aorta, so that they cannot spread across its mouth, and an opening is then left between the valves, in the centre of the vessel, through which the blood flows freely back into the ventricle.—*Vid.* Plate I, Fig. 3.

4*th*.—The valves without any proper organic lesion may be rendered inadequate to their function by dilatation of the mouth of the aorta. The aorta, affected by aneurism, or dilated, as it frequently is in elderly persons, about its arch, will sometimes have the dilatation extending to the mouth of the vessel, and in such a case, the valves become inadequate to their function, not from any disease in themselves, but from the mouth of the aorta dilating to such a diameter, as to render the valves unable to meet in its centre; the blood then, as in the other instances, regurgitates freely into the ventricle.

General Symptoms.—On the general symptoms that accompany this disease, little is necessary to be said. Like most of those connected with affections of the respiratory and circulating organs, they are uncertain and unsatisfactory. There are frequently convulsive fits of coughing, more or less dyspnoea, sense of straitness and oppression across the chest, palpitations after exercise, sounds of rushing in the ears, and inability to lie down. Neither one nor all of these symptoms are essential to the disease. They may all arise from varied affections of the lungs, heart, liver, or nervous system. They neither tell us the seat of the disease, nor the extent of the danger.

Signs.—What is deficient in general symptoms from their obscurity, is, however, amply supplied by the certainty of the physical and stethoscopic signs, which may be referred to the three following indications: 1*st*, Visible pulsation of the arteries of the head and superior extremities. 2*d*, *Bruit de soufflet* in the ascending aorta, in the carotids, and subclavians. 3*d*, *Bruit de soufflet* and *frémissement*, or a peculiar rushing thrill felt by the finger, in the carotids and subclavians. In conjunction with these may be reckoned the pulse, which is invariably full. When a patient affected by the disease is stripped, the arterial trunks of the head, neck, and superior extremities immediately catch the eye by their singular pulsation. At each diastole the subclavian, carotid, temporal, brachial, and in some cases even the palmar arteries, are suddenly thrown from their bed, bounding up under the skin. The pulsations of these arteries may be observed in a healthy person through a considerable portion of their tract, and become still more marked after exercise or exertion; but in the disease now under consideration, the degree to which the vessels are thrown out is excessive. Though a moment before unmarked, they are at each pulsation thrown out on the surface in the strongest relief. From its singular and striking appearance, the name of *visible pulsation* is given to this beating of the arteries. It is accompanied with *bruit de soufflet* in the ascending aorta, carotids, and subclavians, and in the carotids and subclavians, where they can be ex-

amined by the finger, there is felt *frémissement*, or the peculiar rushing thrill, accompanying with *bruit de soufflet* each diastole of these vessels These three signs are so intimately connected with the pathological causes of the disease, and arise so directly from the mechanical inadequacy of the valves, that they afford unerring indications of the nature of the disease In order to understand their value, it is necessary to consider their connection with the cause by which they are produced The visible pulsations of the arteries of the neck, etc may be first examined

In the perfect state of the mechanism at the mouth of the aorta, the semilunar valves, immediately after each contraction of the ventricle, are thrown back across the mouth of the aorta by the pressure of the blood beyond them, and when adequate to their function of closing the mouth of this vessel, they retain in the aorta the blood sent in from the ventricle, thus keeping the aorta and larger vessels distended These vessels consequently preserve nearly the same bulk during their systole and diastole But when the semilunar valves, from any of the causes enumerated, become incapable of closing the mouth of the aorta, then after each contraction of the ventricle, a portion of the blood just sent into the aorta, greater or less, according to the degree of the inadequacy of the valves, returns back into the ventricle Hence the ascending aorta and arteries arising from it, pouring back a portion of their contained blood, become, after each contraction of the ventricle, flaccid* or lessened in their diameter While they are in this state, the ventricle again contracts and impels quickly into these vessels a quantity of blood, which suddenly and greatly dilates them The *diastole* of these vessels is thus marked by so sudden and so great an increase of size as to present the visible pulsation which constitutes one of the signs of the disease

That this visible pulsation of the arteries is owing to the mechanical cause here assigned is made evident by several circumstances It is most distinct in the arteries of the head and neck, which empty themselves most easily into the aorta, and of course, into the ventricle In the arteries of the lower extremities of even larger size than those which present it about the head and neck, it is not seen to any comparative degree, and most generally not at all while the patient is standing or sitting It is much more marked in the arteries of the head and neck in the erect than in the horizontal posture, and a patient suffering under the disease himself, first pointed out a circumstance which is convincing of its being produced as asserted He could increase the pulsation of the brachial and palmar arteries in a most striking degree by merely elevating his arms to a perpendicular position above his head He thus enabled the brachial and palmar arteries to empty themselves more easily back upon the aorta They became more flaccid, and

*It may be objected to the phrase *flaccid*, that the arteries, being capable of contracting upon whatever quantity of blood they may contain are never flaccid In using the phrase, it is not meant that the sides of the arteries, like a collapsed vein fall together but merely that having become emptied of some of their blood in consequence of its regurgitation into the ventricle they are, while in this state, less tense than when at the next diastole they are distended by a fresh supply of blood to their limit of extension

then, on the next contraction of the ventricle, their diastole became comparatively greater, and their visible pulsation of course more marked The same effect could be produced in the arteries of the lower extremities by lying down and elevating the legs on an inclined plane The strength of the heart has little to do in producing this singular pulsation, for it is never observed in an equal degree, and most generally not at all, in the arteries of the lower extremities

If it be asked, is the explanation here adduced of the cause of this visible pulsation sufficient to account for its appearance in the brachial and radial arteries, since the blood to return back from these vessels into the arch of the aorta should flow upward when the patient holds his arms in the ordinary position, flexed or by hanging by his side? The following reply may be made When the subclavians are pouring back their blood into the arch of the aorta and ventricle, the elasticity of the brachial arteries, acting upon the blood just urged into them, forces it back along with the retrograde current of the subclavians, no obstacle meeting it in that direction The brachial arteries thus partially empty themselves, and become in their systole of a lessened diameter like the carotids and subclavians, but in less degree The next jet of blood from the ventricle dilates them, and as in the subclavians, produces in them a visible pulsation, and if they be assisted in returning their blood by elevating the arms to a perpendicular position, their pulsation becomes, as has been already observed, much more strongly marked The arteries of the lower extremities are not similarly circumstanced The arteries of the upper extremities are assisted in emptying themselves back towards the heart, by the retrograde current in the subclavians and ascending aorta, but on the blood contained in the arteries of the lower extremities, the tall column of blood in the descending aorta is pressing, and prevents any return, or if it be supposed that of the large mass of blood in the descending aorta, a small portion flows back into the arch, it can produce little change in the contents of the iliacs and femorals, and moreover, whether the column of blood in the aorta be lessened or not in diameter, the pressure on the contained blood of the iliacs and femorals will remain the same, and keep these vessels distended If we, however, as already observed, alter the relation of the several arteries to the arch of the aorta, so as to facilitate the reflux of their contained blood, for instance from the radial arteries, by raising the arms to a perpendicular line above the head, from the iliacs and femorals, by placing the patient in a recumbent posture, and raising the legs upwards on an inclined plane, the visible pulsation becomes much more marked in these respective arteries

The *bruit de soufflet,* which is heard in the ascending aorta, carotids, and subclavians, with the accompanying *frémissement* in the latter arteries, is next to be considered The *bruit de soufflet* characterizing this disease, is heard, as already observed, in the ascending aorta, its arch, and in the carotids and subclavians It can be followed upwards from the fourth

rib along the course of the aorta, increasing in loudness as it ascends, until it is heard of great intensity at the upper part of the sternum, where the arch of the aorta most nearly approaches this bone, and then branching to the right and left, it can be traced into the carotids and subclavians of both sides, and in these trunks it assumes a harshness that it did not possess in the aorta. This *bruit de soufflet* is synchronous with the visible pulsation, with the diastole of the arteries. It is no consequence whether the ascending aorta and its large branches be sound or be diseased, the *bruit de soufflet* is as loud in the one case as in the other. To account for the presence of this sign, and why it extends so far from the seat of the disease and along sound vessels, it is necessary to refer to a paper published in the *Lancet* of 1829, Vol II, p 1.* Continued observations from the date of that paper to the present, have confirmed the view then taken of the cause of that singular sound, of its being dependent, purely on a physical cause, on a mechanical change in the manner of the blood's flowing.

In that paper is related an experiment, which it may be well to recapitulate here. A flexible tube, such as a piece of small intestine, or a portion of artery, is connected by one end with a tube which has a current of water of considerable force running through it. While the piece of intestine or artery is kept fully distended by the supply of water from the tube, no sound is produced by the motion of the fluid, but if the flexible tube, while the fluid is moving through it, be pressed upon in any part, so that the quantity of fluid passing through the contracted part is no longer sufficient to keep the further portion of the tube tense, then, beyond the contracted part, where the tube is less tense, or in some degree flaccid, a distinct, and, according to the velocity or force of the current, a loud *bruit de soufflet* is heard, and, at the same time, if the finger be gently laid upon the part of the tube where the *bruit de soufflet* is heard, a slight trembling of the tube is perceived, evidently arising from the vibrations into which the current within is throwing its sides. If, in place of constricting any one part of the flexible tube, the whole tract of tube be allowed to become partially flaccid, by diminishing the supply of fluid, and the fluid be then allowed to rush along the tube by jets, at each jet the tube is suddenly distended, resembling the visible pulsation described above, and with each diastole of the tube, there is a sudden and loud *bruit de soufflet,* and, synchronous with the *bruit de soufflet,* there is *frémissement* felt by the finger.

Both the sound heard and the sensation felt by the finger in this experiment may be explained by the principles which regulate the motion of fluids. It may be remarked, that it is a property of fluid in motion, that, when discharging itself from the orifice of a tube into open space, or into a vessel of wider capacity not fully distended, its particles move in lines from the orifice, like so many *radii* tending to leave vacuums between them. When the flexible tube, artery or intestine, therefore, is kept fully dis-

*Corrigan, D. J. Aneurism of the aorta; singular pulsation of the arteries, necessity of the employment of the stethoscope, Lancet 1: 586-590, 1829.—F. A. W., 1940

tended, the fluid moves forward as a mass, there is no tendency in its particles to separate from one another,—they all press equally,—there is no vibratory motion of the sides of the tube, and consequently no sound, and no *frémissement* or trembling But if the tube be not kept fully distended, then the fluid propelled through it rushes along as a current, and its particles tending to leave vacuums between them, throw the sides of the tube into vibrations, which can be very distinctly felt by the finger, and which give to the ear the peculiar sound *bruit de soufflet,* and to the touch *frémissement*

These principles may be applied to the state of the ascending aorta and its branches in the instances before us When the aortic valves are fully adequate to their function of perfectly closing the mouth of the aorta, and thus preventing any regurgitation of blood, the aorta and its branches are kept fully distended, the blood is at each contraction of the ventricle propelled forward *en masse,* and there is no trembling, or vibratory motion of the sides of the aorta, carotids, and subclavians, and, as in the flexible tube when fully distended, no sound is emitted But when the valves, becoming inadequate to their office, permit some of the blood contained in the ascending aorta, carotids, and subclavians, to return into the left ventricle after each contraction, then the aorta and these trunks become, like the flexible tube in the second part of the experiment partially flaccid, and at the next contraction of the ventricle, the blood propelled into them is sent along as a rushing current, which throws the sides of these arteries into vibrations, and these vibrations give to the ear *bruit de soufflet,* and to the finger *frémissement* These two signs may be traced to a varying distance from the mouth of the aorta, and always along the carotids, and to the outer third of the subclavians, and sometimes in the brachial arteries, as far as the bend of the arms, the distance to which they are heard being determined by the limit to which the current-like motion of the blood producing them is extended In those cases in which the deficiency of the valves is considerable, allowing a full stream of blood to rush back into the ventricle, there is heard in the ascending aorta a double *bruit,* the first accompanying the *diastole* of the artery, the second immediately succeeding, and, in listening to the two sounds constituting this double *bruit de soufflet,* the impression made distinctly on the ear is that the first sound is from a rushing of blood up the aorta, the second from a rushing of it back into the ventricle It is impossible for those who have not heard this double *bruit* to conceive the distinctness with which the impression described is made on the ear A patient in one instance heard this double sound distinctly in his own person, and referred it to its cause, a rushing of blood *from* and *to* the heart The *bruit de soufflet* and *frémissement* are not perceived in the arteries of the lower extremities, when the patient is in a sitting or standing posture The pressure of the blood in the abdominal aorta is sufficient in these postures to keep the vessels arising from it fully dis-

tended, and thus no vibratory motion of their parietes being permitted, there is no bellows sound, nor *frémissement* or rushing thrill.

History and Progress of the Disease.—Of eleven cases of the disease, only two occurred in females, and in both of these the valves were nearly quite sound in texture, but the aorta being thinned and dilated, the valves could not meet so as to prevent regurgitation. None of the cases occurred in very early age. The youngest person presented labouring under the disease was twenty years of age. In this respect, inadequacy of the aortic valve differs from narrowing of the left auriculo-ventricular opening, which is not unfrequently met with in children, and even in infants at the breast. The causes of the disease are uncertain. In one case the disease followed an attack of acute rheumatism, which had been accompanied with symptoms of *pericarditis*. In some cases the commencement of the disease was referred by the patient to an inflammatory affection of the chest, which had occurred months or years before, while in others no cause or date could be assigned.

The symptoms accompanying its commencement and progress are very variable. Most generally the patient describes the first sensations as having been a feeling of oppression and straitness across the chest, with palpitation of the heart on any unusual exercise. These symptoms become gradually more distressing, and are after a very uncertain period of time accompanied by fits of coughing resembling paroxysms of asthma, and terminating in scanty expectoration. In a few cases, however, cough was not at any time, even up to the last hours of life, an urgent symptom, the oppression and straitness of the chest, with palpitation on any exertion, and an anxiety for a supply of fresh air, being the principal complaints. As the disease proceeds, the straitness and oppression about the chest become more distressing, fits of coughing more frequent, and the patient has an anxiety, approaching to agony, for a free supply of fresh air, frequently starting from bed at night under the dread of suffocation. In the last stage the state of suffering is extreme. The patient will not lie down for a moment from the dread of suffocation. The face, which had been pale, becomes purple on the lips as in suffocative catarrh, oedema of the legs comes on, followed ultimately by oedema of the hands and arms, there is no sleep, or there are almost incessant startings from it, the countenance assumes a most painful expression of sinking, and the patient at length dies exhausted. The pulse in no case was under eighty. It ranged from that to 110, and in every case it has been all through the disease (unless influenced by medicine) full and vibrating, even to within a few hours of death. In the course of the disease, the superficial branches of the carotids, the brachial arteries, the radial, and the ulnar, and their branches, wherever near enough to the surface to be traced, become apparently enlarged, and remarkably tortuous,—the brachial artery in parts of its course often almost doubling upon itself. The *frémissement* or rushing thrill, described as easily felt in the subclavians

and carotids, can sometimes be felt by moderate tact as fast as the pulse in the wrist The heart in all the cases that occurred was enormously enlarged, and its bulk arose from the state of the left ventricle, which in some cases was so much enlarged in cavity and in thickness, as to make the organ resemble rather the heart of a bullock than that of a man The other parts of the heart, although necessarily obliged to keep pace in some measure with this increased size, did not at all partake equally in the enlarged bulk The impulse of the heart was far less than natural, even in cases where the hypertrophy of the left ventricle was greatest In some of them no impulse could be felt, and in none did the impulse during life give at all a proportional measure of the excessive hypertrophy discovered after death*

Haemoptysis very rarely occurs in the course of the disease, and the lungs are generally found after death permeable to air, and remarkably healthy This is owing to the sound state of the auriculo-ventricular opening of its valves This opening being full sized, permits the blood to pass with freedom into the ventricle where it is retained by the sound auriculo-ventricular valves, and thus those sudden congestions of the blood vessels of the lungs, so common in narrowing of the left auriculo-ventricular opening, are remarkably rare The manner of death in inadequacy of the aortic valves is different from that in narrowing of the auriculo-ventricular opening In the latter, owing to the obstacle presented by the narrowed opening to the passage of blood into the ventricle, the lungs are by any slight exciting cause suddenly congested, and the patient dies, not from the direct effect of the organic affection of the heart, but from the superinduced affection of the lungs,—pulmonary apoplexy, pneumonia, or suffocative catarrh In the disease under consideration, the patient appears to die of mere exhaustion The inefficiency of the valves of the aorta throws a great increase of labour on the left ventricle The muscular energy of this part of the heart is in the course of time worn out The heart is at length incapable of sustaining the column of blood incessantly pressing upon it, it ceases to contract and is found after death largely distended with blood The symptoms preceding death are in accordance with this state For some days, or even

*Laennec has stated and his assertion is supported by many that the degree of impulse is always a correct index of hypertrophy of the ventricle but it is now admitted by some most capable of judging that the impulse of the heart is not to be considered a gauge of the hypertrophy of the ventricle Andral in his *Clinique Medicale*, Vol II p 160 says Plus d une fois dans des cas ou après la mort nous avons *trouve les parois des ventricules très epaissies en meme temps que leurs cavités etaient notablement agrandies, nous n'avions reconnu pendant la vie aucune espèce d'impulsion* Dans d autres cas où il y avait simple hypertrophie due ventricule gauche avec grande diminution de sa cavité (hypertrophie concentrique de MM Bertin et Bouillaud) il n y avait pas eu non plus d impulsion appréciable ' Piorry (*Sur la Percussion* p 139) says that impulse of the heart carried even to raise the head of the observer is far from being a constant sign of hypertrophy Dr Graves in a clinical lecture (*vid* Med Gazette March 1831 p 714) says I can assert in the most positive manner, that I have seen cases of pneumonia in which the heart s pulsation continued violent until within a short time of dissolution so much so indeed, as to induce the erroneous belief in myself and other medical attendants, that this organ was in a state of hypertrophy and dilatation and yet it was found after death to be in every respect healthy ' This subject will be resumed at another time

weeks, before death, nature appears to be struggling against overwhelming exhaustion The patient is constantly in the most heart-rending tone imploring to be relieved of the weight that is upon him, the countenance expresses the greatest sinking and distress, there are anxious calls for fresh air and a continual restlessness, similar to what is seen in a patient sinking from hemorrhage, and when in this state the patient in some trifling motion dies exhausted

The duration of this disease is very uncertain No case was of less duration than two or three years, and some of the cases at present under treatment have been of seven or eight years standing The time during which the disease may continue without terminating fatally, seems to depend principally upon the extent to which regurgitation is permitted The cases in which the valves, from small perforations, allowed but little regurgitation continued for many years, while the case which furnished the Plate No II and in which the valves were ruptured and much injured, allowing considerable regurgitation, terminated fatally in less than three years

Diagnosis—Inadequacy of the aortic valves may be confounded with narrowing of the mouth of the aorta, either congenital or from diseased valves, with disease of the auriculo-ventricular valves, with aneurism of the arch of the aorta or *arteria innominata,* with nervous palpitations, and with asthma Congenital narrowing of the mouth of the aorta is a very rare disease, but narrowing of the mouth of this vessel produced by vegetations on the valves is not unusual, and *bruit de soufflet* is a sign common to it, and to the disease we are considering The resemblance between the signs of the two diseases extends, however, no farther The visible pulsation of the arteries, arising from the arch of the aorta, which forms so striking a sign of inadequacy of the aortic valves, is wanting in narrowing of the mouth of the aorta The pulse also is strikingly different in the two diseases In narrowing of the aortic orifice it is small and contracted, in inadequacy of the aortic valves it is invariably full and swelling In narrowing of the aortic orifice there is generally a marked contrast between the pulse and the impulse of the heart The pulse is small and contracted, the impulse of the heart is strong and energetic In the disease we are considering, when there is a contrast it is always in the inverse way, for while the arteries beat with violence, and the pulse is strong and full, the impulse of the heart is scarcely perceptible When the mitral valves, becoming indurated or ossified, produce narrowing of the auriculo-ventricular opening, that narrowing produces *bruit de soufflet,* and the *bruit de*

*The principles regulating the motion of fluids, already laid down explain the production of *bruit de soufflet* in narrowed auriculo-ventricular opening The blood at each contraction of the auricle discharges itself from a narrow orifice into the ventricle, "a vessel of wider capacity not fully distended"—"The particles of the blood 'move in lines from the orifice like so many radii tending to leave vacuum between them' This motion, as in the experiment of the tube throws the sides of the ventricle into vibrations which produce on the ear *bruit de soufflet,* and if the heart thus affected come forward so as to transmit through the parietes of the chest this vibrating motion the hand laid over the heart perceives a *fremissement,* or trembling in the organ, the *bruissement* of Corvisart

pulsation arising from inadequate aortic valves, and in females these palpitations will last not only for months but for years, and seem to justify an opinion that there is organic disease of the heart This nervous palpitation is not, however, accompanied by *bruit de soufflet* and *fremissement*, and the absence of these two signs is conclusive as to the nature of the disease Sometimes, however, more than one examination is required before pronouncing a positive opinion, for in a nervous patient, the alarm excited by the first examination will render the circulation hurried and irregular, and hence there may be in the carotid or subclavian a momentary *bruit de soufflet* In making the examination it is moreover necessary, that the edge of the stethoscope should not be allowed to press on the artery, because its pressure is sometimes sufficient in those cases to produce the sound When the *bruit de soufflet* and *fremissement* are only momentary, no value should be attached to them In permanent patency of the aorta they are never absent The convulsive fits of coughing ending in difficult mucous expectoration have made some cases of this disease be mistaken for asthma, and the state of the pulse has served to maintain the error, for the pulse being remarkably full, as it always is in the disease we are considering, seemed to be sufficient evidence that there was in the heart no obstruction to the circulation, hence the convulsive fits of coughing were supposed to have their origin in the lungs With a knowledge of the signs afforded by the disease, no one of even moderate acquaintance with the stethoscope can confound it with asthma, without a knowledge of the stethoscope it will, however, be impossible in very many instances to distinguish between the two diseases General symptoms will give no information on which the slightest reliance can be placed

Treatment —There is no class of diseases to which the scientific principles that guide modern medicine have been less applied than to diseases of the heart From its curious mechanism, from the varied derangements to which that mechanism is subject, from the number of tissues that enter into its formation, and from its numerous sympathies, its diseases frequently demand most opposite lines of treatment, and yet, it would seem, from the perusal of works on the subject, that one principle were thought sufficient for guiding the treatment of nearly all the diseases of this important organ With the idea of heart disease, is too frequently associated the notion that such disease, without regard to its precise nature or its cause, requires the action and continued enforcement of measures calculated to exhaust strength and depress vital energy, and this error is sanctioned by the standard works on the treatment of heart disease

Corvisart says, that "in a great number of organic lesions of the heart, as, for example, in active aneurism, the indication is to diminish the general strength of the patient, and that of the heart in particular " Laennec, p 739, says that "though we cannot remove indurations of the valves and narrowing of their openings, we are nevertheless in such

cases to follow up the same measures (bleeding and starving), to remove or diminish hypertrophy," and Bertin, p 233, states that "the treatment of valvular alterations is to consist of general and local bleedings, of low diet, of preparations of digitalis," etc , and, p 367, "that the measures to be employed against hypertrophy are to be essentially antiphlogistic, and calculated to produce debility " A little reflection on the nature of the disease now before us will show that these principles are inapplicable both to the treatment of the valvular alterations, and of the hypertrophy of the left ventricle, which accompanies that alteration

The disease we are considering is an inadequacy in the valvular apparatus at the mouth of the aorta permitting a regurgitation of blood into the ventricle In the perfect state of the valvular apparatus at the mouth of the aorta, the valves support by intervals the column of blood in the aorta, and the heart with its ordinary complement of fibre and of muscular strength, is with this assistance competent to the office it has to perform But when, in consequence of a deficiency in the valvular apparatus, the heart does not receive its due share of assistance from these valves, and is obliged to perform not only its own function of propelling the blood, but has in addition to support after each contraction a portion of that weight of blood which should then be wholly supported by the valves, it is no longer in its ordinary state equal to the task imposed upon it In such circumstances, nature, to enable the heart to perform the additional labour thrown on it, increases its strength by an addition of muscular fibre, and the heart thus becomes hypertrophied, in accordance with the general law, that muscular fibres become thickened and strengthened when there is additional power required from it Is this hypertrophy disease, or is it a wise provision of nature, by which the organ is thus made equal to the increased labour it has to perform? On the answer depends the treatment to be adopted, and on this there is no room for hesitation A heart of ordinary strength could not, under the circumstances, carry on the circulation, and nature then wisely endows the heart with the requisite degree of strength It is at once obvious that to interfere with this wise provision of nature, to diminish the strength of the heart, or, if we choose other words, to direct, *according to the advice of Laennec, Bertin, etc* our measures against the hypertrophy of the organ, is to deprive the system of the only power which enables the heart to carry on the circulation No one thinks of directing measures to diminish hypertrophy of the muscular tissue of the stomach, in narrowing of the pylorus from scirrhus of the bladder or rectum in stricture of the urethra or intestines

In these instances the hypertrophy is recognized as a provision of nature to make the power of the part equal to the obstacle it has to overcome, and yet, this simple principle seems to have been entirely overlooked in diseases of the heart, as if this organ possessed muscular fibres

of a different nature from other organs, or as if, in adapting itself to obstacles affecting its action, it follows laws different from other muscular parts. The consequence of the neglect of this principle has been, that too often, in treatment of a valvular alteration in the heart, there has been a constant struggle between nature and medicine. Nature has been making the organ equal to its task, while medicine has been directed to counteract nature's efforts, and, by weakening the organ, to render it totally incapable of its task. The repeated bleedings, the starvings, the enforcement of debilitating measures, are totally unsuited to the disease we are considering.

Instead of such treatment, the measures most beneficial are those which by strengthening the general constitution, will give a proportionate degree of vigour to the muscular power of the heart, and thus enable it to carry on the circulation in the absence of that assistance which it ought to receive. With this view, a generous and sufficient diet of animal and vegetable food should be advised, at the same time that an abstinence from those beverages, such as malt liquors, which increase much the mass of the fluids, should be enjoined. It is not at all necessary that the patient should be prohibited from attending to his business or profession, provided that he do not devote to it so much attention as to produce debility. And as there is among patients who have learned that they are afflicted with heart disease an universal dread of sudden death, it is necessary to undeceive them on this point, and in the present instance it can be done with perfect safety, as the termination of the disease is never sudden. This plan of treatment, opposite to what has been generally enjoined, was forced upon the attention long before the reasoning adduced here had been brought to support it.

One case may be mentioned, out of many that occurred, showing the bad effects of debilitating treatment in the disease before us, and exemplifying the evil of acting as if one principle were sufficient for guiding us in the treatment of all heart diseases. It is now several years since a consultation was held upon the case alluded to. This treatment ordered was in accordance with that generally recommended, consisting of repeated small bleedings, blistering, the exhibition of digitalis, and the most rigid regulation of diet, a total abstinence from animal food, and even a spare allowance of vegetables and milk. At the time the patient, a young man, was put under this treatment, he was not in an alarming state, but the disease being recognized as heart disease, he had the fortitude to submit to a course which he was led to expect held out a prospect of cure. Bleeding after bleeding, and blister after blister, were repeated, starvation enforced, and digitalis exhibited, until the patient was reduced to such weakness that he had scarcely strength to raise himself in bed. The local disease was all this time, however, growing worse, for the

palpitation, cough, etc, were, from the slightest cause, increased to greater violence than previously to the commencement of treatment The plan was, nevertheless, persevered in, until the patient's death being supposed at hand, this debilitating treatment was discontinued From that hour the patient got better, and as muscular strength returned, the embarrassment of the breathing, palpitation, cough, etc, became less and less urgent The patient is still alive, the disease is still present, but, with full living and good air, he is able not only to take considerable exercise, but even to undergo the fatigue of a business that constantly requires very laborious exertion

Having laid down the plan of treatment proper to be adopted as far as it produces effects upon the system, and through it upon the heart constituting a part of the system, it now remains to examine the propriety of employing in this disease a remedy such as *digitalis,* which produces a specific effect upon the heart, rendering its action slow and weak, and which in consequence of that effect is usually recommended in cases of heart disease in conjunction with the measures already deprecated In inadequacy of the aortic valves the pulse generally ranges from 90 to 110 After each contraction of the ventricle during the pause or interval of rest occurring between that contraction and the next following, a quantity of blood is regurgitating into the ventricle The danger of the disease is in proportion to the quantity of blood that regurgitates, and the quantity that regurgitates will be large in proportion to the degree of inadequacy of the valves, and to the length of pause between the contractions of the ventricle during which the blood can be pouring back If the action of the heart be rendered very slow, the pause after each contraction will be long, and consequently the regurgitation of blood must be considerable Frequent action of the heart, on the contrary, makes the pause after each contraction short, and in proportion as the pauses are shortened, the regurgitation must be lessened Instead, then, of regarding an increase of frequency in the action of the heart as an aggravation of the disease, it must be viewed, as we have already viewed hypertrophy of the heart, as a provision for remedying as far as possible the evil consequences arising from inadequate valves To retard in such circumstances the action of the heart would be to do an injury In every case of this disease in which *digitalis* has been administered, it has invariably aggravated the patient's sufferings The oppression has become greater, the action of the heart more laboured, the pulse intermittent, and very often dicrotic, from the heart's being unable by a single contraction to empty itself, general congestion and dropsy, if present, have been increased, and in some of the instances *bronchitis* from congestion has been induced, the respiration became laborious, and the strength so much sunk, that patients seemed almost moribund From this state they only recovered by omitting the *digitalis,*

and putting them on stimulants. In no case of this disease did *digitalis* produce the slightest good effect, and in all, the patients while under its exhibition were always worse.

A *moderately quick pulse indeed is of itself no evil,—it is only an evil* as an indication of some disease. In the present instance it is, however, an index of a positive good, it shows that the pauses between the contractions of the ventricle are short, and, consequently, that there is less danger of the quantity of blood thrown back upon the ventricle in the pause of its action, or intervals of rest, being of any considerable amount. The pulse, which in this disease ranges from 90 to 100, or even rises higher, is not to be interfered with merely because it is more frequent than natural. The more frequent action of the ventricle indicated by that pulse is a safeguard against regurgitation. In this respect permanent patency of the aortic opening differs from narrowing of the auriculo-ventricular opening. In permanent patency, as already explained, frequent contractions and short pauses are the best safeguard against regurgitation. In narrowing of the auriculo-ventricular opening, on the contrary, slow action of the heart is an object of the first importance, for a slow action of the auricle will allow more time for the passage of the blood through the narrowed opening, and thus diminish the regurgitation upon the pulmonary veins and the lungs.

Although depleting measures and a lowering plan of treatment have been deprecated in the management of inadequacy of the aortic valves, and although the continued employment of such measures in cases of this disease instead of bringing relief, never fails to produce an aggravation of symptoms, there are, however, circumstances in which the most active treatment is called for. Inflammatory affections, congestions, etc., are more likely to occur in patients suffering from any obstacle to the circulation than in others, and whenever these supervene in patients labouring under inadequacy of the aortic valves, whether they be inflammatory affections or congestions of the thoracic or abdominal viscera, they call for the most prompt and active treatment. Instead of the heart affection inducing us to be less active in our treatment, it is a motive to be more prompt than in ordinary cases. It may be a fatal mistake to suppose that the presence of the heart disease is to make our measures less energetic. Pneumonia, peritonitis, acute rheumatism, have occurred in some of the cases that have been under observation, and the patients have borne depletion even better than persons without any heart affection. It is a curious fact that, bleeding carried to a very large amount has never in these cases produced fainting. In this case the well-established principle, that in acute diseases the more prompt and decided the measures, the more rapid will be the recovery of the patient, and the less troublesome the sequelae of the disease, should be our guide. The inflammatory affections that may in-

cidentally occur in cases of inadequacy of the aortic valves, should be opposed with promptitude and decision; bleeding, when used, should be large; but when the inflammatory affection is once subdued, we should cease as soon as possible from debilitating treatment,—which, if persevered in, will prove injurious to the organic affection. No details need be given of the measures to be adopted in those incidental affections, because they differ in no respect, except in energy from the usual treatment for the affection, whatever it may be, that has chanced to supervene. This active treatment on the occurrence of local inflammation is not at all incompatible with the course recommended to be followed when there is no disease present but the valvular inadequacy. Those incidental inflammatory or congestive affections will be rendered less likely to occur by the previous course of management recommended; for the greater vigour the system has enjoyed, the less danger there is of slight causes producing inflammation or congestion.

Lastly, there is besides the supervention of local inflammation or congestion, yet another circumstance, in which blood-letting may be required. Without the occurrence of any apparently adequate cause, straitness of chest, difficulty of breathing, tumultuous action of the heart, and a general feeling of nervous oppression are complained of. Neither pneumonia nor inflammatory action in any organ can be detected, and these symptoms seem to arise from an increase of bulk in the absolute mass of blood circulating, which keeps all the vessels so distended that the heart becomes oppressed, incapable of freely contracting, and tumultuous in its action. Relief is at once afforded by a large blood-letting, speedily followed by the exhibition of a full dose of an opiate. For the employment of the opiate, I am indebted to the suggestion of my colleague, Dr Hunt, and its good effects are such as to have no substitute for it. The employment of a large bleeding in either of the circumstances here detailed, is very different from the repetition of those irritating small bleedings that are usually practised.

Fits of coughing are sometimes very troublesome in the course of the disease, and where they arise from trifling bronchitis, they are best relieved by pectoral mixtures with a large proportion of opium, not less than four or five grains to an eight ounce mixture.

In these observations no medicine or treatment has been recommended with the view of acting directly on the aorta or valves, so as to restore in any degree the function of the latter. There is no medicine that can have any such power after the disease has been of much standing; and if the valves have become perforated or broken, it is obviously impossible to restore them to their original state. The disease is seldom seen in the commencement. Perhaps if seen early in those cases where it has followed an attack of rheumatism or where it partakes of an inflammatory

character, the employment of mercury pushed to salivation, and counter-irritation, might check the progress of the disease. In the advanced stages no good effect on the valvular affection has been produced by any or all of these measures.

Although the cure of *Inadequacy* of the Aortic Valves is probably out of the reach of medicine, a correct knowledge of the nature of the affection is not the less necessary. The patient is relieved from harrassing treatment, that, however, applicable in other cases of heart disease, is not alone useless, but positively injurious in this. In other affections of the heart there is a constant danger of sudden death from pulmonary apoplexy or hemorrhage, which may be induced even by ordinary exertion, and such danger keeps the patients in a state of perpetual terror. In this disease, on the contrary, assurance may be given against any sudden termination, and the patient may be permitted not only to attend to his business or profession, but may be assured, that, in leading a life of business and tolerable activity, he is adopting the very best means to prolong his life. Under treatment such as recommended, it is astonishing what little uneasiness inadequacy of the aortic valves will produce,—indeed, very often not so much as those organic affections or growth of the liver, which are nevertheless viewed by the profession and by patients with much less terror.

13, Bachelor's Walk, Dublin.

1835

JEAN BAPTISTE BOUILLAUD

THE PATHOLOGY OF ENDOCARDITIS

JEAN BAPTISTE BOUILLAUD

Portrait by C H Lehman, 1875

(Courtesy Charles C Thomas)

JEAN BAPTISTE BOUILLAUD

(1796 1881)

ON SEPTEMBER 16, 1796, Jean Baptiste Bouillaud was born at Bragette, near Angoulême in France He attended the Lycée d'Angoulême and distinguished himself by winning the "prix d'excellence" He also received a prize at this school for a poem written in Latin

Encouraged by his uncle, a surgeon-major in the French army, Bouillaud decided to become a member of the medical profession He therefore left his native town to study medicine at Paris His studies were interrupted by the march of the Allies on Paris Part of Napoleon's army under Marshal Auguste Marmont (1774-1852) and Marshal Édouard Mortier (1768-1835) unsuccessfully attempted to withstand the Allies Bouillaud joined the students of the École Polytechnique in this ineffectual resistance Later, when Napoleon returned from Elba, Bouillaud, who had great admiration for the Corsican, enlisted in a Hussar regiment After the defeat of Waterloo, Bouillaud resumed his medical studies

It was Bouillaud's good fortune to have many illustrious teachers Among the most prominent were Guillaume Dupuytren (1777-1835), whom Bouillaud attended in the master's final illness Dupuytren left special instructions that Bouillaud should perform the necropsy His other teachers included François Joseph Broussais (1772 1838), of whose blood-letting theories, unfortunately, Bouillaud was a fervent disciple, Jean Nicolas Corvisart (1755-1821), under whom, no doubt, Bouillaud developed an interest in cardiology, and François Magendie (1783-1855), who inspired Bouillaud with an interest in experimental physiology

In 1818, Bouillaud became an intern in the hospitals of Paris, and in 1823 he received the degree of Doctor of Medicine After his graduation he became an intern at the Hôpital Cochin under René-Joseph-Hyacinthe Bertin (1757-1828) There he assisted his chief in Bertin's work on diseases of the heart, which was published in 1824

In 1825, at the early age of twenty-nine, according to Lereboullet, he was named a member of the Académie de Médecine and a year later he received the degree of "agrégé," and became assistant professor

Bouillaud succeeded Joseph Récamier (1774-1852) in 1831 as professor of clinical medicine of the Faculté de Médecine of Paris The cholera epidemic occurred during that year and he had occasion to observe many patients suffering from cholera at the Hôpital de la Pitié, where he was a member of the staff In 1832 he published his observations on cholera and attributed cures to local bleeding combined with cauterization of the spinal column He also denied the contagious nature of the disease

Some time later Bouillaud became a member of the staff at La Charité in Paris In 1835 he published his "Traité clinique des maladies du coeur" In this work is recorded his interesting description of endocarditis, to which he applied the name, and we are presenting to our readers, in translation, his observations on the pathologic aspects of this disease Trousseau, according to Rolleston, suggested that the term "maladie de Bouillaud" should be given to endocarditis "which was an almost unknown affection until the illustrious professor of the Hôpital de la Charité drew the attention of the medical world to it in a description to which nothing could be added"

Bouillaud also contributed to cardiology his "law of coincidence," which states:[1] "In the great majority of cases of acute generalized febrile articular rheumatism, there exists a variable degree of rheumatism of the fibrous tissue of the heart. This coincidence is the rule, and the non-coincidence the exception."

In the second edition of his treatise on the heart (1841) Bouillaud devoted considerable space to a study of the measurements of the heart, and, according to Rolleston, these were the first accurate methods of weighing and measuring this organ.

Bouillaud also described certain physical signs connected with the cardiovascular system. These included his "bruit de diable," the venous humming sound heard over the internal jugular vein in chlorosis, the "bruit de rappel," the false reduplication of the second sound heard at the apex and characteristic of mitral stenosis, and a tinkling sound sometimes heard on the right side of the apex beat in hypertrophy of the heart. Potain, as we shall later show, credited Bouillaud with being the first to describe gallop rhythm.[2]

Bouillaud is also remembered for his pioneer work in neurology. His most important contribution in this field was his identification of the anterior lobes of the brain as the center of speech. Although he highly esteemed the work of Franz Gall (1758-1828), Bouillaud in 1827 published two papers[3, 4] refuting Gall's opinions regarding the function of the cerebellum. Gall thought the cerebellum to be an organ of the instinct of propagation. Bouillaud gave evidence that the cerebellum was the organ of equilibration, station, and progression. He also demonstrated that the lesions of the cerebellum affected co-ordinate movements.

According to Rolleston, Bouillaud is also to be credited with making an advance in the study of acute articular rheumatism, which in France is often referred to as "maladie de Bouillaud."

In 1846, Bouillaud published his last important work: "Traité de Nosographie Médicale." This was an extensive study in five volumes setting forth, in considerable detail, his elaborate doctrines.

Bouillaud was elected a member of the Chambre des Députés for Angoulême in 1840. In 1848, he became dean of the Faculté de Médecine of Paris. In 1862 he was elected president of the Académie de Médecine, and in 1867 he served as president of the first International Medical Congress, which was held in Paris. In 1868, Bouillaud was made a commander of the Legion of Honor.

Bouillaud was at first opposed to the ideas of Pasteur, but later, in 1879, acknowledged the value of his work. He also was extremely critical of the salicylic treatment of rheumatism introduced by Germain Sée in 1877.

Bouillaud's death took place on October 29, 1881, in his eighty-sixth year. Four years later his statue, the work of Verlet, was unveiled at Angoulême in the presence of several of his friends and pupils, including Velpeau, Laboulbène, Cornil, and Potain.

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[1] Rolleston, J. D.: Jean Baptiste Bouillaud. Proc. Roy. Soc. Med. (Sect. Hist. Med.) 24: 1253-1262, 1931.

[2] See page 531.

[3] Bouillaud, J.: Recherches expérimentales tendant à prouver que le cervelet préside aux actes de la station et de la progression, et non à l'instinct de la propagation. Arch. gén. de méd. 15: 64-91, 1827.

[4] Bouillaud, J.: Recherches cliniques tendant à réfuter l'opinion de M. Gall sur les fonctions du cervelet, et à prouver que cet organe préside aux actes de l'équilibration, de la station et de la progression. Arch. gén. de méd. 15: 225-247, 1827.

TRAITÉ CLINIQUE

DES

MALADIES DU CŒUR

PRÉCÉDÉ DE

RECHERCHES NOUVELLES SUR L'ANATOMIE
ET LA PHYSIOLOGIE DE CET ORGANE;

PAR J BOUILLAUD,

PROFESSEUR DE CLINIQUE MÉDICALE A LA FACULTÉ DE MÉDECINE DE PARIS

AVEC DES PLANCHES GRAVÉES.

> Scripsi illa, quæ sensuum testimonio inter labores et tædia iterum iterùmque expertus sum.
>
> (AVENBRUGGER)

TOME PREMIER.

PARIS,

J.-B. BAILLIÈRE,

LIBRAIRE DE L'ACADÉMIE ROYALE DE MÉDECINE,

RUE DE L'ÉCOLE-DE-MÉDECINE, N 13 BIS,

LONDRES, MEME MAISON, 219, REGENT-STREET

1835.

ON THE PATHOLOGY OF ENDOCARDITIS*

By

JEAN-BAPTISTE BOUILLAUD

EXPOSITION AND APPRECIATION OF THE ANATOMICAL CHARACTERISTICS OF ENDOCARDITIS

THE anatomical characteristics of this inflammation, essentially the same as those of pericarditis, offer, however, very remarkable peculiarities depending on the one hand upon the structure of the parts affected by the endocarditis and on the other, upon the presence of blood which circulates incessantly through the cavities lined by this membrane. It is of the greatest importance to know thoroughly the anatomic characteristics proper to each one of the periods of endocarditis, if one would comprehend in a clear and precise manner the principal symptoms which it produces in its course. Thus, in order to have as complete an idea as possible of these characteristics, it is necessary to study successively the alterations of the sero-fibrous tissue itself, the products produced by the inflammatory reaction, and the state of the blood contained in the cavities of the heart. After having thus considered in all their phases the anatomic lesions of which endocarditis may be the source, we shall take care to compare them with the results of inflammations in other organs, and we shall reply to the objections which have been made against our method of deriving the origin of certain organic lesions of the heart.

DESCRIPTION OF THE ANATOMIC ALTERATIONS

I. *First Period of Endocarditis (Period of Sanguinary Congestion, of Softening, and of Ulceration or Suppuration)*

1. The redness of the endocardium is one of the anatomical characteristics the study of which must occupy us in the first place. This redness has been noted in 12 or 13 patients reported earlier. The absence of this redness in one of our patients does not astonish us, since it is known that in cases in which a membranous inflammation leads rapidly to death it may occur that one finds no redness in the cadaver, although this redness may have been present during life. To the innumerable facts of this nature already observed we may add case 5 of our series, in which a marked erysipelatous redness was replaced in the cadaver by a pallor similar to those areas of skin which had not been inflamed

**Traité Clinique des Maladies du Coeur,* Paris 1835 pp 170-192. Section Deuxième, Histoire Générale de l'Endocardite. Article Premier. Translated by Erich Hausner, M.D., Amsterdam, New York.

Nevertheless, in the immense majority of cases, acute endocarditis leaves in its track a redness more or less marked; this color is sometimes rosy and sometimes scarlet, occasionally violet, poppy colored or even brownish. It is partial or general; it often involves only the valves, and almost constantly when it is present in the entire endocardium it is of maximum intensity in the valvular portion of this membrane. Ordinarily, the redness is deeper in the right heart than in the left, which is probably due, at least in part, to the fact that the blood which circulates in the former is less deeply red and less vividly red than that which circulates through the latter. However that may be, this redness is not due to a capillary injection, but to a sort of sanguinary tint of the internal membrane of the heart. Ordinarily it does not penetrate beneath this; it does not disappear on washing, but it does not resist a maceration sufficiently prolonged.

The nature of the redness of the internal membrane of the heart and vessels has been recently the object of numerous discussions. [*Here follows a quotation from Laennec on this subject.*]

It is difficult to make more researches than I already have on this anatomico-pathologic subject treated here. But, it results from these researches that this redness of the internal membrane of the heart cannot be considered other than as one of the results of the inflammation of this membrane. I am convinced, on the other hand, by a large number of facts, that certain rednesses of the heart and of the vessels are nothing but purely cadaveric imbibition, and I have recognized, with many other observers, that these latter rednesses are almost constant in individuals opened at a period when putrefaction of the body is more or less advanced, above all when the individual has succumbed to an illness accompanied by putrid or typhoid phenomena, cases in which the blood is more liquid than normal, which renders the internal membrane of the vascular system more likely to imbibe.

I do not believe that it is possible to decide by simple inspection, nor by washing or by maceration, whether a given redness of the internal membrane of the heart is the effect of an inflammation or of a cadaveric imbibition. It is necessary to search elsewhere to find the means to solve the grave questions which we examine here. To my way of thinking, one may regard as being of inflammatory nature any redness of the internal membrane of the heart existing in any individual whose body has been opened before any trace of decomposition has been noted, and which during life had presented symptoms which we shall assign in the following article to inflammation of the internal membrane of the heart. But the inflammatory nature of the redness will be more certain if to the conditions which I have already mentioned be added the following: (1) swelling, thickening of the reddened parts; (2) the presence of a certain quantity of pus, pseudomembranous matter or even of adherent dis-

*Translator

colored clots or fibro-albuminous specks, (3) the coincidence of a similar redness in vessels in which one has positively diagnosed the inflammation before death

The observations which we have reported in our preceding paragraphs bring together all, or most, conditions under which a redness is to be attributed to inflammation

A notable thickening of the internal membrane of the heart often accompanies the inflammatory redness above mentioned, when the endocarditis has lasted a certain time, twelve, fifteen, twenty days and more, for example Nevertheless, this thickening does exist in a marked fashion except over the valves, where the membrane is in a manner double and fortified by fibrous tissue The fungous swelling of these valves has been reported in many of our cases

The softening of the internal membrane of the heart is not always evident in this period It has seemed to me, however, that this membrane breaks with more facility than the normal membrane At the same time, the surface was a little less polished than the normal, and more or less wrinkled The cellular tissue beneath the endocardium appeared also, in these cases, to have lost its force of cohesion, and to have become fragile, and because of this to become detached from the endocardium

One observed sometimes in the acute period of endocarditis erosions of commencing ulcerations of the internal surface of the heart, or of the valves themselves (see Case 50) These ulcerations may become the origin of perforations of the wall of the heart, of the valves, or of the interventricular or intraventricular septa

2 A purulent or pseudomembranous secretion certainly takes place in endocarditis, but one can imagine that it is often difficult to prove the existence of it In fact, such is the rapidity and force of the blood flow through the chambers of the heart, that the product secreted little by little by the inflamed endocardium, must be incessantly swept away by this current Nevertheless, in certain cases a small quantity of true pus or pseudomembranous material is encountered following an acute endocarditis The pus is sometimes hidden in the centre of a clot or in the net formed by the columnae carnae The same is true of pseudomembranous material This latter, of great tenacity, adheres solidly to the parts where it is deposited, and one finds portions of it on the surfaces of the valves, on their free borders, and on their tendons, where it sometimes presents itself under the form of granulations or globules of albuminous consistency or semi-solid fibrin In certain cases, it is true, one may mistake a simple adherent, discolored, elastic clot for a pseudomembranous production The error is the more easy to make since pseudomembranous products themselves are formed from solid fibrin, and the error is fundamentally unimportant

Can an acute endocarditis terminate in gangrene? In reflecting on the extreme rarity of such a termination in membranes which, like the endocardium, are serous, one is naturally inclined to answer this question in the negative. Nevertheless, I have gathered four or five cases in which I have been tempted to believe that it was an internal inflammation of the heart, strongly analogous to certain gangrenous phlegmasias, to which one must attribute the very rapidly fatal termination that took place. The observations made in Cases 22 and 39 appear to me to belong to this category. *[Here the author briefly discusses two cases, one post-traumatic (gas gangrene?) and the other with disordered heart action and high fever which had a reddening of the endocardium at autopsy without ulceration and in one of which bubbles of gas occurred in the very liquid blood in the right heart, which he prefers not to believe was due to post-mortem changes.]**

To return to the subject of gangrenous endocarditis or malignant, as the ancients would have called it, I think that this is a subject to review, and which merits serious researches.

3. We have just passed in review the principal lesions which are presented by an inflamed endocardium, under the double point of view of its structure and its secretory function. Nothing remains for us but to make known the lesions of the blood that is found in the cavities of the heart. But, numerous observations reported in our preceding chapters have indicated that this phlegmasia commonly carries with it the coagulation of a greater or lesser quantity of blood which circulates through the chambers of the heart. In this regard, endocarditis behaves itself in the same way as arteritis and phlebitis. The sanguinary concretions formed under the influence of an acute endocarditis must not be confounded with the ordinary clots found in the heart, especially with those which develop after death. The concretions consecutive to acute endocarditis are white, colorless, elastic, glutinous, adherent to the walls of the heart, wound around the valvular tendons strongly analogous to pseudomembranes, some of these at times present points or red lines which are nothing but the rudiments of vessels.

The concretions which we have examined differ markedly in respect to volume and configuration. They ordinarily are prolonged into the great vessels. Everything being equal, they are more voluminous, more abundant in the right heart than in the left. Their maximum adherence in general occurs near the free edges of the valves, where one finds fragments after repeated washings. (It is probable that these little fibrinous masses may organize and transform themselves into vegetations or granulations.)

II. *Second Period of Endocarditis (Period of Organization of Secreted Products or of a Portion of Fibrinous Concretions)*

*Translator's note.

When endocarditis does not terminate in prompt resolution, if it is prolonged a considerable time (fifteen, twenty, thirty days or more), the inflamed tissues are more or less thickened and the plastic part of the products abnormally secreted by these tissues pass from an amorphous state into a state of organization. Then, according to accidents of position, of configuration or of composition of the organizable matter, one encounters either vegetations or granulations, either cellulo-fibrinous adhesions or fibrinous or serofibrinous layers, and so forth.

The vegetations or granulations have a remarkable predilection for the valves and particularly for their free border. Also, one finds them in certain cases on the internal surface of the cavities of the heart and especially the auricles. (Observation XLVII of *Traite de l'auscultation mediate* offers a remarkable example.) They have been divided by M. Laennec into two divisions: globular vegetations and verrucous vegetations. The first appear to us more exactly described as albuminous or fibrinous granulations. The expression "verrucous" is happily chosen, for the vegetations or excrescences to which they apply resemble warts. One may thus designate them under the denomination of "cornified" or "cartilaginous" vegetations or excrescences.

Do these two kinds of vegetations, granulations or excrescences, represent essentially different entities or are they simply two manifestations of a single accidental production? Do some come from the organization of a portion of solidified fibrin and the others from the organization of a true pseudomembranous exudation? Before considering their mode of generation let us begin by describing them in detail.

The albuminous or fibrinous vegetations (*globuleuses* of M. Laennec) are soft, easily crushed, like too solid albumin or a half-organized fragment of fibrinous pseudomembrane. They may be detached by a light traction. Their color is grayish white or yellow, mixed sometimes with a rosy tint, or definitely red. These granulations have appeared to me to be analogous to those which are sometimes found on the surface of the chronically inflamed pleura, the pericardium or the peritoneum; the resemblance is even so striking that one would have difficulty in distinguishing them, as I have assured myself by comparing the granulations of the pleura with valvular vegetations, both from the same subject.

The verrucous vegetations, very analogous to venereal warts, are held and implanted so firmly, and are rooted with such tenacity, that they may almost be said to form an identity with the tissue to which they are attached. The tissue of these vegetations is, as it were, cornified; it makes a noise under the instrument which divides it like fibrocartilage.

The number of either albuminous or verrucous vegetations is very variable; the same is true of their size and configuration. Some are no larger than a millet seed, others are the size of flax seed or a small pea.

*See page 362.—F. A. Willius 1940.

They are sometimes isolated, scattered, discrete, as it were, while others are united in groups, confluent, or so disposed as to imitate cabbage flowers. Their form is often rounded, spherical, but often elongated, cylindrical or flattened. Their surface is sometimes smooth and polished and sometimes unequal and irregular.

It is rare that these vegetations of the valves or of the internal wall of the heart exist alone; usually, as our observations show, they are accompanied by a fibrocartilaginous or calcareous induration of the valves. However this may be, however they are multiplied, confluent, grouped like cabbage flowers, they give rise to a narrowing of the orifices to which are adapted the valves which they overload and whose movements they impede; the existence of this peculiar type of narrowing is an important circumstance to note.

Let us return to the method of production or the pathogenesis of these vegetations. [*Here the author quotes M. Laennec to the effect that vegetations are probably formed as a consequence of some trouble in the circulation and are polypiform or fibrinous concretions formed through absorption and nutrition analogous to those processes which convert an albuminous false membrane into cellular tissue; also the resemblance between these processes and the crystallizations which form when a thread is placed in a saturated saline solution.*]* †

This comparison seems very ingenious to me and I do not doubt that fibrin organized on the edge of valves may organize into vegetations. But this is no reason to deny the role that endocarditis plays in the production of these vegetations, for as we have already demonstrated by positive facts, this phlegmasia determines the formations of organizable fibrinous concretions. M. Laennec is thus wrong in opposing our opinion, in saying that "if the inflammation of the internal membrane of the heart was the efficient cause of the vegetations in question, they should have as origin and common ancestor a false membrane extending like a layer over the valves, which does not occur."

But if we admit that fibrin, solidified under the influence of endocarditis or under any other influence, if you will, may be transformed into vegetations, it will be permissible to admit also that pseudomembranous matter, secreted by the inflamed endocardium, may like fibrin itself of which it is really a modification, dispose itself so as to constitute small rounded masses which organize themselves little by little into vegetations. Such a doctrine is the more legitimate, since the vegetations, similar to those developing on the interior surface of the heart, also develop on the serous exterior of the same organ, as also on the surface of the pleura and of the peritoneum in certain cases of pseudomembranous inflammations of these membranes. But, in these cases, one cannot attribute the vegetations observed to polypiform concretions.

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*Translator's note.

†See page 362.—F. A. Willius, 1940.

But again, in supposing that the vegetations of the internal membrane of the heart always had their origin from a polypiform concretion, we should not be less authorized to place it among the accidental effects of endocarditis since, by the statement of M Laennec himself, it is one of the sanguinary concretions due to inflammation It remains only to determine in which cases the polypiform concretions which may give rise to vegetations should have been really consecutive to a generalized or partial endocarditis But we have limited ourselves to report here cases of vegetations coinciding with definite traces of an old endocarditis

The adhesions that endocarditis may leave in its wake have not been mentioned by any of the authors of the *traités sur les maladies du coeur* I have reported six observations in this work One can easily conceive why adhesions should be less frequent in endocarditis than in inflammations of other serous membranes The torrent of blood which flows through the cavities of the heart, the movements of the valves, are powerful obstacles to the formation of these structures of which we speak Nevertheless, in spite of these obstacles, adhesions sometimes form, and as we might foresee, these adhesions are formed at points where the above mentioned obstacles are at a minimum Thus, we have encountered them between the less movable leaflets of the valves and on the corresponding wall of the ventricles

These adhesions are in general very firm The cases which we have cited show their usual disposition Such adhesions inevitably cause difficulty in the circulation, since they do not permit the valves completely to close the orifice to which they are adapted

There is another type of adhesion of the valves which is more common than the preceding, notably that which establishes itself between the opposing borders of the valvular leaflets in certain cases of narrowing of the orifices, which we will describe below

As a consequence of the adhesions of the endocardium, one must mention the organized false membranes which cover more or less of the surface There are such which cover nearly the whole area of the cavities of the heart One may even find many layers organized, one above the other

Instead of false membranes as extensive as the above, one finds them sometimes hardly more than four, five or six lines in diameter, which I designate under the name of *spots* of the endocardium They are similar to the milky spots of the pericardium, and also to certain white spots of the cornea, whose name I must give them One can lift them easily with forceps, underneath one finds the endocardium intact or only a little more opaque than normal

In a great number of cases, the thickening of the internal membrane of the heart is due to the presence of organized false membranes which we have just mentioned Also, there are other cases in which the endocardium is

truly thickened, hypertrophied, and then has lost its transparence, and the surface has become less polished, and wrinkled, unequal and a little villous

The hypertrophic thickening of which we speak extends also to the fibrous tissue as well as to the subendocardium cellular tissue I have found frequently hypertrophy of the valves and of the tendinous zone where their adherent border is inserted The bicuspid valve is above all subject to this consecutive hypertrophy, and if there also occurs a slight narrowing of the orifice, one sees on the side of the auricle a wrinkling of the adherent border of the valve, which we will describe later

III *Third Period (Period of Cartilaginous, Osseous or Calcareous Induration of the Endocardium in General, and of the Valves in Particular, With or Without Narrowing of the Orifices of the Heart)*

We have seen in the preceding chapters the internal membrane of the heart and valves thicken and hypertrophy and the products secreted through the influence of the inflammation organize into cellulo-fibrous, fibrous or even fibro-cartilaginous tissue (The tissue of verrucous vegetations belongs to the latter category) It remains to us to study the cartilaginous, osseous or calcareous productions which remain as a consequence of an endocarditis

The productions considered here present themselves under diverse forms Sometimes they are simple circumscribed points of the size of a lentil, sometimes sheets or plaques of the size of a fingernail or a piece of money of 10 *sous* or larger, sometimes more or less rounded masses The entire valves are sometimes converted into cartilaginous or osseous tissue Also, the fibrous zone of the orifices of the heart and the points of the valves are the parts affected by preference by cartilaginous degeneration Between the plaques or incrustations of the valves, one finds spaces in which the tissue is normal or presents only a slight degree of hypertrophy

The osseous or calcareous incrustations of the valves have the most varied configuration Some are bent in an arc, or in a hoop, others are elevated and elongated in the form of stalactites, of pyramidal or irregular form, still others are rounded into spheres and resemble true calcareous concretions of which the volume is sometimes that of a pigeon's egg or even of a small hen's egg These kinds of calculi or stones developing on the surface or in the substance of the valves, are ordinarily covered with sharp points, or inequalities on their surfaces, and simulate the calculi designated as *muraux* (mural calculi) These ossifications or petrifactions sometimes send prolongations into the substance of the heart

The chondrified, ossified or petrified valves show the most varied changes in their conformation When the accidental productions of which they are the seat consist of simple points or small blades, the thickened and slightly rigid valves conserve still their mobility, and can rise and fall as in the normal state, but when the organized valves are more profoundly altered,

when the degeneration has invaded them in all of their parts, they become more or less completely unable to perform the functions that nature has confided to them. In certain cases their leaflets are folded or rolled upon themselves, and then they represent straight ribbons; they then are too short and too immobile to be able to close the orifice to which they are adapted. The *insufficiency* of the valves is evident in this kind of deformation; the orifice may be dilated, which renders their insufficiency greater. The valvular leaflets may be perforated or torn, at the same time that they are indurated and thickened; in one case one of the valves of the aorta, almost entirely detached, hung and floated, so to speak, in the cavity of the aorta.

In a great number of cases, the valves affected with induration and thickening are united and attached by their neighboring edges, and from a sort of membrane or diaphragm pierced in its center by a narrow opening, sometimes rounded, sometimes oval or elliptic. The thickening and induration sometimes invade the chordae tendinae and columnae carneae.

The large number of observations that we have reported give an idea of the principal variations in form, extent and aspect that the productions that we are studying may produce.

The narrowing of the orifices of the heart consecutive to the different kinds of indurations and valvular transformations merits a detailed description. In giving this description we shall have occasion to designate a few other dispositions of the valves which must be studied with the narrowing itself.

The narrowing of the orifices of the heart, following different kinds of thickening and induration of the valves, is the gravest anatomic accident that endocarditis may carry in its wake. In order to appreciate this, one must recall the normal dimensions of the orifices of the heart, as they have been established in our *Prolegomenons*.

The degrees of narrowing of the orifices of the heart are very variable. In extreme degrees, one can hardly introduce the tip of the little fingers, or even the tip of a writing pen, into the narrowed orifice.

The opening which is left between the thick, indurated valves or those valves united by their neighboring borders, is permanent, or constantly open. It is sometimes rounded, oval or elliptic. It resembles in many cases a buttonhole or a glottis of which the lips are represented by the rounded borders of the thickened valve leaflets. This comparison applies particularly to certain narrowings of the left auriculo-ventricular orifice. In a few cases, the leaflets of the bicuspid valve have acquired an enormous thickening and protruded from the side of the auricle; then the retracted orifice may be compared to the orifice of the cervix uteri, and like this imitates a fish mouth. Seen from the auricular side, the circumference of the retracted auriculo-ventricular orifice presents a very pronounced folding, as if this circumference had been folded upon itself,

this disposition gives it the appearance of the external circumference of the anus or the opening of a purse drawn together with cords

In general, in cases with a considerable narrowing of the orifices, the valves, rendered immobile by their rigidity, are elevated so as to cross the axis of the orifices almost at a right angle Also, they are often enough reversed in the direction of the blood flow Then the orifice represents a sort of infundibuliform canal of which the summit presents to the ventricles, if the auriculo-ventricular orifices are the site of narrowing, and toward the aorta and pulmonary artery, if the narrowing affects the aortic or pulmonary orifices Nevertheless, as for the aortic valves, one observes an inverse disposition, that is to say, they are dejected toward the ventricular cavity

To give an exact idea of the principal forms that the narrowed orifices of the heart may show, and to emphasize the peculiarities which distinguish the narrowing of each one of these orifices, we have believed it wise to reproduce here, in a note, the description of the principal cases of this kind of lesion, which we have reported in the first section of this chapter [*A note gives the pathologic description of hearts extracted from case reports by the author, reported in the first part of the book*]*

We will not terminate this article without remarking to the reader that this narrowing which is so common in the orifices of the heart, as a consequence of a prolonged chronic endocarditis, is a new fact of resemblance between this phlegmasia and those which affect other hollow organs What physician is ignorant that the urethra, the neck of the bladder, the arteries, the excretory canals for the tears, saliva, bile, the different regions of the digestive tube, and especially the cardia, the pylorus, the junction of the ileum with the cecum, the inferior portion of the rectum etc , etc , what physician, I say, is ignorant that all these parts just mentioned may undergo a narrowing of greater or lesser degree, as a result of a long and slow inflammation? It is not possible here to mention the results which follow these mechanical lesions, the gravity of which is proportionate to the importance of the function to which they impose a more or less invincible obstacle

However that may be, the preceding details are sufficient to bring clearly to light this truth to which we have called the attention of the reader in treating the diseases of the heart in general, that the diverse lesions of that organ engender reciprocally one another, and that lesions of nutrition or of organic or vital action, for example, always result, when they are not arrested or strangled in their cradle, so to speak, by producing physical or mechanical lesions which are too often resistant to all measures known to our art

*Translator's note

1846

WILLIAM STOKES

DESCRIPTION OF HEART BLOCK

WILLIAM STOKES

(Courtesy Medical Classics)

WILLIAM STOKES

(1804-1878)

"We have to do with something which cannot be measured or weighed, something, too, in which experiment can only be used within narrow bounds, an element whose nature is yet unknown, fleeting in its action, and every day producing new combinations, not merely new because they were never observed before, but really new as appearing for the first time."

—William Stokes on medical education

WILLIAM STOKES, the second son of Whitley Stokes, was born in Dublin. His distinguished father was a physician and Regius professor of medicine in the University of Dublin, and in 1800 was appointed professor of medicine in the Royal College of Surgeons at Dublin.

William Stokes was tutored by the well-known scholar, John Walker, who taught him the classics and mathematics. Some time later, Stokes studied clinical medicine in Meath Hospital. He learned the auxiliary sciences at both Trinity College and the Royal College of Surgeons at Dublin. He also spent two years in Glasgow, where he worked in chemistry under the direction of Professor Thompson. From Glasgow, Stokes went to Edinburgh, where he completed the required studies for the medical degree.

At Edinburgh it was Stokes's good fortune to be the pupil of William Alison, professor of medicine. And it was the stimulation received from Alison which led Stokes at an early date to achieve prominence among the pioneers of medical science.

At about this time the medical profession was much concerned with diagnosis of diseases of the chest, a concern that was brought about by the delayed acceptance of Auenbrugger's system of percussion and the development of auscultation by Laennec. William Stokes, shortly before he was graduated from medical school, published the first systematic work in the English language on the use of the stethoscope,[1] "An Introduction to the Use of the Stethoscope," printed in Edinburgh in 1825 by Machlachlan and Stewart. This work and two subsequent lectures,[2] published in 1828, formed the basis for his more mature account of diseases of the chest which he published in 1837.

After Stokes had been graduated from Edinburgh (1825), he returned to Dublin to become physician to the Dublin General Dispensary. In 1826, at the age of twenty-two, he succeeded his father as physician to the Meath Hospital. At Meath he had as a colleague the distinguished Robert Graves, who became his lifelong friend. Stokes and Graves did much at Meath to improve the system of clinical teaching.

In April, 1828, Stokes was married to Miss Mary Black, to whom he had been engaged for three years.

Following Graves's prediction of the event in 1826, Asiatic cholera broke out in Ireland at some time in 1832. Stokes and Runley reported the first case of cholera

[1]Dr Cullen to whom Stokes had dedicated his work and Sir J Forbes had earlier published reports of cases illustrating the practical use of the stethoscope.

[2]*Two Lectures on the Application of the Stethoscope to the Diagnosis and Treatment of Thoracic Disease*, Dublin 1828 Hodges and McArthur.

in the Dublin epidemic, an achievement which, as it turned out, entailed considerable personal risk The two physicians had been sent to inquire into the cause of a certain mysterious death which had occurred at Kingstown After their inspection, they pronounced the deceased person to have died of Asiatic cholera of the worst type The crowd which had gathered outside the house of the deceased person received the announcement calmly at first, then, realizing the horror of the situation, invoked its wrath on the physicians Members of the mob hurled stones and sticks at them Stokes and Runley escaped in their carriage, but it was well battered when they reached home

Stokes's first work of major importance was his treatise on diseases of the chest,[3] published, as we have mentioned, in 1837 This book did much to elucidate the phenomena of thoracic disease In his book Stokes followed the line of investigation initiated by Corvisart and Laennec According to Corrigan[4] Stokes's volume added much to the work of Laennec Stokes, moreover, clarified an issue which the followers of Laennec had neglected to elucidate Stokes pointed out that in diagnosis, physical signs must be associated with symptoms

Among the important observations found in Stokes's book were (1) the discovery of a stage of pneumonia prior to that described by Laennec as the first stage, (2) the discovery of a displacement of the heart as the result of the rapid absorption of pleuritic effusion in the right side, and (3) the employment of the stethoscope as an aid to the detection of foreign bodies in the air passages

After the publication of Stokes's work on thoracic disease, he received many honors The University of Dublin granted him the degree of Medicinae Doctor, honoris causa, he was elected a fellow of the King's and Queen's College of Physicians in Ireland, he was made an honorary member of the Imperial Academy of Medicine of Vienna, and of the royal medical societies of Berlin, Leipzic, Edinburgh, and Ghent His fame had even spread to the United States, where he was elected to the National Institute of Philadelphia

Such a favorable reception of his work provided a healthy stimulus to Stokes, both in his writing and in his clinical teaching During the next decade his attention was drawn chiefly to the diseases of the heart and he contributed many papers on this subject to the Dublin "Quarterly Journal of Medical Science" He was an editor of this journal, in association with Robert Graves and William Parker, from 1836 until 1842 The articles he contributed to it formed the basis for another work equally important to his earlier work on thoracic diseases This was "The Diseases of the Heart and the Aorta," published in 1854 at Dublin by Hodges and Smith

From the chapters of the aforementioned work we have reprinted his classic description of "Cheyne-Stokes respiration" in connection with fatty degeneration of the heart[5] This type of respiration had been noted earlier by John Cheyne, but Cheyne did not associate any diagnostic importance with the syndrome Stokes also referred in his book on the heart to his memoir on slow pulse[6] Therein he had described the condition first noted by Adams which is now called "heart block with the Adams-Stokes syndrome" This passage, also, we are reprinting

Stokes in his book on the heart also advocated pursuance of a system of graduated muscular exercises to aid in the removal of cardiac debility, especially among younger persons The book is additionally famous for its accurate descriptions of pericarditis, valvular diseases, and weakening of the heart in typhus fever

[3]*A Treatise on the Diagnosis and Treatment of Diseases of the Chest*, Part I, *Diseases of the Lung and Windpipe* (no more published), Dublin, 1837 Hodges and Smith

[4]Quoted by Stokes's son in *William Stokes, His Life and Work (1804-1878)*, London, 1898, T F Unwin, p 65

[5]See pp 484-489

[6]This was first published in the Dublin Quarterly Journal of Medical Science 2 73-85 1846

In 1861, the honorary degree, ''Legum Doctor,'' was conferred upon Stokes by the University of Edinburgh In 1865 the University of Oxford conferred on him the same degree Stokes was further honored in 1867 by election to the presidency of the British Medical Association The annual meeting of the association was held that year in Dublin Through Stokes's efforts, graduate education in state medicine was established at Dublin Oxford and Cambridge soon followed in this respect

Stokes was elected to the presidency of the Royal Irish Academy in 1874 Failing health did not permit him to hold this office for more than two years In that same year (1874) Stokes received the honorary degree, ''Legum Doctor,'' from the University of Cambridge During this time Foley was working on a statue of Stokes It was unveiled in 1876 and now stands in the hall of the Royal College of Physicians in Dublin

In 1876 Stokes was presented with the Prussian order, ''Pour la mérite,'' originated by Frederick the Great He was one of the few physicians ever to receive this honor On a professional visit that same year he was injured by a fall from a car This accident was followed by symptoms of spinal concussion, and it seemed to be the cause of the development of the paralytic affliction which gradually weakened him and finally deprived him of the use of his limbs Early in November, 1877, he suffered a sudden paralytic seizure from which he never rallied On January 6, 1878, he quietly passed away

OBSERVATIONS ON SOME CASES OF PERMANENTLY SLOW PULSE[*†]

By

WILLIAM STOKES, M.D

Physician to the Meath Hospital

IN THE fourth volume of the Dublin Hospital Reports, Mr. Adams has recorded a case of permanently slow pulse, in which the patient suffered from repeated cerebral attacks of an apoplectic nature, though not followed by paralysis. The attention of subsequent writers on diseases of the heart, has not been sufficiently directed to this case, which is an example of a very curious and, as there is reason to believe, special combination of symptoms. The following cases will still further elucidate a subject on which there is but little information extant:—

Case I. Repeated pseudo-apoplectic attacks, not followed by paralysis, slow pulse, with valvular murmur.

Edmund Butler, aged sixty-eight, was admitted into the Meath Hospital, Feb. 9th, 1846. He stated that his health had been robust, until about three years ago, at which time he was suddenly seized with a fainting fit, in which he would have fallen if he had not been supported. This occurred several times during the day, and always left him without any unpleasant effects. Since that time he has never been free from these attacks for any considerable length of time, and has had, at least, fifty such seizures. The fits are very uncertain as to the period of their invasion, and very irregular as to their intensity, some being much milder and of shorter duration than others. They are induced by any circumstance tending to impede or oppress the heart's action, such as sudden exertion, distended stomach, or constipated bowels. There is little warning given of the approaching attack. He feels, he says, a lump first in the stomach, which passes up through the right side of the neck into the head, where it seems to explode and pass away with a loud noise resembling thunder, by which he is stupified. This is often accompanied by a fluttering sensation about the heart. He never was convulsed or frothed at the mouth during the fit, but has occasionally injured his tongue. The duration of the attack is seldom more than four or five minutes, but sometimes less, but during that time is perfectly insensible. He never suffered unpleasant effects after the fits, nor had anything like

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*Published in the Dublin Quarterly Journal of Medical Science 2: 73-85, 1846. We reprint from Medical Classics 3: 727-738, 1939.—F. A. W. 1940.

†Stokes's account of 'Fatty Degeneration of the Heart' is reprinted on pp. 484-489.

paralysis. His last fit occurred about one month before admission. He has never heard it remarked that there was anything peculiar about his heart or pulse. At first he found that spirits was the best restorative or prophylactic, but lately he has not used them, being "afraid to die with spirits in his belly."

On admission, he was haggard and emaciated, but seemed the wreck of what was once a fine, robust man. He lay generally in a half drowsy state, but when spoken to was perfectly lively and intelligent.

What he sought admission into hospital for was an injury he had sustained, by a fall, on the left shoulder; this, however, was of no consequence, and he soon recovered under the use of an anodyne liniment.

He makes no complaint of his general health; his appetite is good, and he sleeps well; bowels regular, and, in fact, all the functions are in good order. He has, however, some cough, attended with a slight mucous expectoration. His intellectual powers are perfect. He complains of a feeling of chilliness over the body, and is never warm except when close to the fire. This has long been the case; and he says that each day he gets a periodical chill, generally in the afternoon, which is followed by increased heat of the surface, but without sweating.

On percussion, the chest is universally resonant. The respiratory murmur loud, and combined, more especially posteriorly, with large mucous râles. The impulse of the heart is extremely slow, and of a dull, prolonged, heaving character, giving the idea of feeble as well as of slow action. The first sound is accompanied by a soft bruit de soufflet, which is prolonged until the commencement of the second sound, and is heard very distinctly up along the sternum, and even into the carotid arteries. The second sound is also imperfect, though very slightly so; the imperfection being much more evident after some beats than after others. Pulse twenty-eight in the minute, of a prolonged, sluggish character; the arteries pulsate visibly all over the body, but no bruit is audible in them. They appear to be in a state of permanent distention; the temporal arteries ramifying under the scalp, just as they are seen in a well-injected subject. All the other cavities and viscera appear to be in a perfectly healthy state. Urine, neither acid nor alkaline; of a high colour, clear, specific gravity 1.010; and does not afford a precipitate with nitric acid. He was ordered four ounces of wine, and a liniment for the shoulder.

February 17th. The pulse has varied from twenty-eight to thirty in the minute. The cardiac murmurs continue unchanged; that with the first sound is plainly audible over the upper part of the thorax, but most evident along the course of the aorta.

21st. Pulse thirty. Cough quite gone. Has been complaining of a feeling of the "lump in the stomach" for several days, and was once threatened with the approach of a fit during the night; it passed off, however, without becoming a true attack.

23rd An edematous swelling has appeared behind the left ear, extending up the side of the head, slightly tender on pressure, no redness, has had no shiverings, tongue clean, bowels free Pulse up to 36

March 3rd On the 24th of February the edema had left the left side, and made its appearance on the right, from which it was dispersed on the following day by the application of poultices The pulse fell to the usual range

His aspect and general health are greatly improved since his admission He gets up every day, and is much stronger The shoulder is almost quite well The pulse has continued at about 28 or 30 He says he has had two threatenings of fits since his admission, both occurring in bed, and both warded off by a peculiar manoeuvre, as soon as he perceives symptoms of the approaching attack, he directly turns on his hands and knees, keeping his head low, and by this means, he says, he often averts what otherwise would end in an attack

4th He has mentioned, for the first time today, that he is much troubled with irritability of the bladder, so that he is obliged to rise very often during the night to pass water His urine was examined and found to be healthy Specific gravity 1 015 He has been subject to this for the last twelve months, and it probably depends on the disease of the prostate so common in old men

We remarked today, that on listening attentively to the heart's action, we perceived that there were occasional semi-beats between the regular contractions, very weak, unattended with impulse, and corresponding to a similar state of the pulse, which thus probably amounts to about 36 in the minute, the evident beats being only 28, so that there must be about eight of these semi-beats in the minute, but these signs are very indistinct

14th Health improving, has had no fit, no cough Both morbid sounds are loudest over the sigmoid valves, and thence along the aorta No semi-beats audible Pulse 29, not quite so prolonged as before

18th He complains today of palpitation, and a feeling of uneasiness about the heart,—the impulse is increased and is found to consist of two distinct pulsations The bruit, with the first sound, is somewhat louder than before On listening attentively, there are heard occasional abortive attempts at a contraction, probably about four in the minute They do not destroy the regular intervals between the stronger sounds, but are heard, as it were, filling up the interval We could not recognize a corresponding state of the pulse, which counted 32 in the minute

After this, little change was observed His health continued improved, he had no fit, or threatening of one, and he appeared anxious to leave hospital, in order to go to work again The pulse continued about the same standard, and regular, I believe it never exceeded 36 in the minute

since his admission into the hospital The physical signs remained unchanged, as was observed the day before he left the hospital An examination of the lungs revealed no morbid sign, the bronchial râles, heard at the time of admission, having quite disappeared

He left the hospital in March, intending to go for some time into the country before he resumed work He was advised to be careful not to over-exert himself, and never to allow himself to be bled when threatened with one of his fits

Within the present month (June) this patient has been again admitted into hospital The cardiac phenomena remain as before, but a new symptom has appeared, namely, a very remarkable pulsation in the right jugular vein This is most evident when the patient is lying down The number of the reflex pulsations is difficult to be established, but they are more than double the number of the manifest ventricular contractions About every third pulsation is very strong and sudden, and may be seen at a distance, the remaining waves are much less distinct, and some very minor ones can also be perceived These may possibly correspond with those imperfect contractions which have been already noticed in the heart The appearance of this patient's neck is very singular, and the pulsation of the veins is of a kind which we have never before witnessed

He has had scarcely any of the cardiac attacks since he was discharged, he refers the premonitory sensations to the right supra-clavicular region, but states that he has often experienced them without any loss of consciousness following

The next case exhibits a similar condition of the heart, but the pseudo-apoplectic attacks did not occur

Case II Anemic condition, very slow pulse, with valvular murmur, death, apparently from syncope

A man, upwards of fifty years of age, was admitted, presenting much of the general characteristics of senile phthisis His skin was of a pale yellowish tint and his whole appearance indicated great debility He complained of cough and dyspnea, but did not refer any of his suffering to the region of the heart His pulse was generally 35 in the minute, though occasionally rising to 40 The action of the heart was regular, but feeble, and a valvular murmur with the first sound, precisely similar to that in mitral-valve regurgitation, was always audible This became louder on ascending the sternum, and was most intense on the right side, at the anterior articulation of the second rib We were inclined to consider this as an example of mitral valve disease, and supposed at first that the aortic murmur might result from anemia The patient died without any struggle On dissection, the mitral valve was found healthy The aortic valve was thickened and narrowed, but not permanently patent Water poured into the aorta did not pass into the ventricle, the heart was soft and flabby, and though not an example of complete fatty degeneration,

was covered by a very thick layer of fat The aorta presented several atheromatous patches

In this case the second sound remained normal, there was no regurgitation into the ventricle The valve was sufficiently diseased to cause a murmur with the first sound, but from its power of closing completely, the second remained unaltered

The co-existence of aortic murmur with the symptoms of weakened heart in both these cases is important, for it should appear that this combination is one of frequent occurrence, we shall have less difficulty in recognizing an obscure disease of the heart There is no reason to believe that there is any necessary connection between the weakened, or fatty state of the heart, and disease of the aorta or its valves, but that the combination is frequent appears probable from the following considerations

First—In the two cases which have now been given, we see the combination of slow pulse with aortic murmurs

Secondly—In one of these, organic disease of the aorta was found on dissection

Thirdly—In Mr Adams' case the aortic valves were studded with specks of bone The state of the aorta is not noticed but the carotids and middle arteries of the dura mater presented bony depositions*

Fourthly—In a case published by Dr Cheyne, in the second volume of the *Dublin Hospital Reports*, in which the heart had greatly degenerated into fat, the valves were sound, but the aorta was studded with atheromatous concretions

Fifthly—Professor Law, in his original and important observations on the connection between disease of the heart and brain, in the seventeenth volume of the *Dublin Journal of Medical Science*, gives an account of the appearances observed in examining the body of the Earl of K, and states that the pulse was remarkably infrequent, sometimes not exceeding twenty-five beats in the minute The patient was subject to syncope The examination was made in London, and no mention is made of the state of the muscular substance of the heart, but it was found that the semilunar valves of the aorta were thickened and partially ossified, so that they could not effectually have closed the orifice The brain was extensively softened, and the ventricles distended with a limpid fluid, and the substance of the left hemisphere, both cortical and medullary, was so softened as to present an almost creamy consistence The arteries at the base of the brain presented opaque yellow depositions

This case was, in all probability, an example, if not of fatty degeneration, at least, of a weakened state of the ventricle It is another example of the combination of a singularly slow pulse, tendency to syncope, and disease of the aortic valve

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*See account of Robert Adams page 398

I am indebted to Mr Adams for the particulars of an interesting case of slow pulse, with lesion of the aortic orifice and remarkable softening of the left ventricle The patient had been in excellent health up to within a few months previous to his death He had no palpitation, dyspnoea, nor irregularity of the pulse He had been exposed to various debilitating causes, and, when seen by Mr Adams, presented a slow pulse and visible pulsation of the arteries of the neck The pulse fell to below forty, and a loud bruit de soufflet could be heard along the aorta and in the region of the heart Mr Adams found the heart to be one of the most friable he had ever met with, breaking down under the slightest pressure of the fingers The valves of the aorta were less diseased than could have been expected, considering the state of the pulse, and the visible pulsations noticed in all the arteries The valves were not inadequate to perform their functions, from their being diseased or altered in their structure of form, but the calibre or area of the aorta was so expanded that they could not prevent reflux into the ventricle

I have lately seen another case presenting the combination of a pulse under thirty, repeated pseudo-apoplectic attacks, not followed by paralysis, and distinct valvular murmur with the first sound The gentleman is advanced in life, but enjoys very good general health He has always found that the attacks were increased whenever he was lowered by regimen or medicine He takes a moderate quantity of wine, and is thus able to ward off the malady

The preceding observations go to prove that the combination of the permanently slow pulse, with a diseased condition of the aortic opening, is not uncommon We owe to Dr Corrigan, the important practical observation, that in cases of permanent patency of the aortic valve, the patients do not generally bear a reducing system, but are best treated by a tonic, or even stimulating regimen, and I entirely agree with Professor Law in his opinion, that the pseudo-apoplectic attacks, in cases of slow pulse and weakened left ventricle, are more frequently attributable to a diminished or feeble circulation, than to one of active congestion

We have thus seven cases of permanently slow pulse In five, organic disease of the aorta or the valves, or both, was discovered on dissection, and in four, a manifest aortic murmur existed, in two of the cases the second sound was normal, and in two there was the murmur of regurgitation in the aortic valve

I do not believe, however, that the aortic murmur is any direct sign or necessary combination of the weakened heart Its occurrence in these cases manifestly arises from the combination of aortic disease, and we have abundant evidence that a weakened heart, without aortic disease,

*In Dr Robert Smith s published cases of fatty degeneration of the heart the valves were healthy The patients were both very old women and no stethoscopic observation is recorded The pulse was very slow These patients were not under Dr Smith s care See also the important case of fatty degeneration of the heart communicated to the Pathological Society by Mr Carmichael—Transactions of the Society for 1840

may exist, and yet no murmur be produced. In the typhoid softening of the heart, we have rarely recognized a valvular murmur, and where it did occur, there was reason to believe that carditis had supervened. The typhoid softening, with a pulse from 30 to 40, commonly exists without any murmur.

In Dr. Cheyne's patient a remarkable state of the respiration was observed for some time before death. "For several days," says Dr. Cheyne, "his breathing was irregular; it would entirely cease for a quarter of a minute, then it would become perceptible, though very low, then by degrees it became heaving and quick, and then it would gradually cease again. This revolution in the state of his breathing occupied about a minute, during which there were about thirty acts of respiration."

I once witnessed this condition of breathing, but had not an opportunity of making a dissection. The patient was a gentleman of about sixty years of age, and of spare habit; his ailments commenced with a sudden and severe attack of dyspnea, which subsided, leaving him to all appearances perfectly well; this returned at irregular intervals. When I saw him he had a full, soft, pulse, and a loud though varying murmur with the first sound, propagated into the aorta. At the top of the sternum the murmur was intense, and on several occasions the arteries seemed to pulsate with a force much greater than could be expected from the impulse of the heart. For more than two months before his death, this singular character of respiration was always present, and so long would the periods of suspension be, that his attendants were frequently in doubt whether he was not actually dead. Then a very feeble, indeed barely perceptible inspiration would take place, followed by another somewhat stronger, until at length high heaving, and even violent breathing was established, which would then subside till the next period of suspension. This was frequently a quarter of a minute in duration. I have little doubt that this was a case of weakened and probably fatty heart, with disease of the aorta.

Professor Law has lately communicated to me the following note of a case under his care in Sir Patrick Dun's Hospital. It is an example of a very weak heart, with the pseudo-apoplectic attacks.

Colin Baird, admitted March 10th, 1846, aetat. 44, states that about three years ago he was suddenly attacked with a fit, which he describes as coming on without any warning; his sight suddenly failed, and he fell down; this fit only continued a minute or so, leaving him stupid for some time. After the first attack the patient states that his health was bad for two months, when he resumed his trade, being occasionally attacked at irregular intervals, varying from two to three months, till the attacks became more frequent, and at length came on ten or twelve times in the twenty-four hours. These attacks are induced by smoking, or drinking

spirits The patient, who complained of weakness, appeared worn out, and older than he really is, pulse thirty, heart's action very weak, and the sounds scarcely audible

Dr Law treated the attacks as examples of syncope, and prescribed a generous diet and stimulating medicines On one occasion the pulse fall so low as twenty-four in the minute It was observed, occasionally, that after each distinct beat of the heart there was a species of commotion of the organ, as if another pulsation was attempted These peculiar minor pulsations also occurred in the case of Butler, and were observed on several occasions It would seem as if in the weakened heart, there were two kinds of contractions, in one, the systole of the heart is broken up, as it were, into a number of slight and irregular contractions, giving a permanently irregular and rapid pulse This is the sort of pulse which is so common in the combination of hepatic and cardiac disease, such as occurred in the case of Mr Colles In the second form, the contractions are complete, and with long intervals, and this is seen in the cases now under consideration But the occurrence of these minor pulsations is interesting, as shewing a connecting link between the cases of weakened heart, with a rapid though irregular pulse, and those where the pulse is slow and regular

The preceding observations are published with view of drawing the attention of the Profession to a combination of cerebral and cardiac phenomena, of which our knowledge is still imperfect

1852

WILLIAM SENHOUSE KIRKES

DESCRIPTION OF EMBOLISM FROM INTRACARDIAC COAGULA

WILLIAM SENHOUSE KIRKES

(Courtesy St Bartholomew's Hospital Journal)

WILLIAM SENHOUSE KIRKES

(1823-1864)

WILLIAM SENHOUSE KIRKES, according to Power, was born at Hilker, in North Lancashire, England, in 1823 He received his primary education at the Cartmel Grammar School He then became apprenticed to a firm of surgeons at Lancaster In 1841, Kirkes began his studies at St Bartholomew's Hospital, where he proved to be a brilliant student In 1842 he received the highest grades in chemistry and in 1843, he ranked first in surgery In 1844, he was first in medicine, midwifery, medical jurisprudence, and clinical medicine

In 1846, Kirkes received his degree of Doctor of Medicine from the University of Berlin St Bartholomew's Hospital appointed him medical registrar and demonstrator of morbid anatomy in 1848 In 1850 he became a licentiate of the Royal College of Physicians, and in 1855 he was elected a fellow of that organization In 1856 he delivered the Goulstonian Lecture

Kirkes was appointed assistant physician to St Bartholomew's Hospital in 1854, and physician in 1864, a few months before his untimely death He lectured at St Bartholomew's Hospital on botany, and, with Dr Patrick Black (1813-1879), delivered joint lectures on medicine

The diseases of the vascular system were of special interest to Kirkes, and in 1852, he contributed the first English article on "Embolism from Intracardiac Coagula," in which he confirmed Virchow's views, which had been published a few months earlier Because Kirkes's description of the condition leaves little to be desired, we have included it among our classic accounts

Kirkes expected to write a work on diseases of the heart, but unfortunately, death intervened before he had collected all his material

Dr Kirkes was a member of the commission appointed by the Admiralty and Horse Guards to make an investigation of the problem of venereal disease He had served in this capacity for a short while, when suddenly he was afflicted by pneumonia accompanied by pleurisy and pericarditis The disease made rapid progress and he died on December 8, 1864

Kirkes made a transcription of Sir James Paget's "Lectures on Physiology" which was published in 1848 as the "Handbook of Physiology, by W S Kirkes assisted by James Paget" Paget's name was omitted from subsequent editions of this work, which was published as "Kirkes' Physiology" until the issuance of the fourteenth edition, at which time Halliburton became the author

The friends and former students of Kirkes raised a fund of money by subscription to provide a memorial to him For many years this consisted of a gold medal awarded annually to the student of St Bartholomew's Hospital who passed the best examination in the diagnosis and treatment of patients in the medical service in the wards of the hospital In 1885, Mrs Kirkes provided a fund that caused the aggregate of the annual prize to be thirty pounds, in addition to the medal

ON SOME OF THE PRINCIPAL EFFECTS RESULTING FROM THE DETACHMENT OF FIBRINOUS DEPOSITS FROM THE INTERIOR OF THE HEART, AND THEIR MIXTURE WITH THE CIRCULATING BLOOD*

By

WILLIAM SENHOUSE KIRKES, M.D.

Licentiate of the Royal College of Physicians, Registrar and Demonstrator of Morbid Anatomy at St Bartholomew's Hospital

THAT the fibrinous principle of the blood may, under certain circumstances separate from the circulating fluid during life, and be deposited within the vascular system, especially on the valves of the heart, is a fact so clearly established and so generally admitted, that I need only, at the outset of the communication I have the honour to present to this Society, allude to it as a settled truth, and refer, for the proofs, to the various general works on diseases of the heart and blood-vessels, and to such special essays on the subject as those of Dr Burrows[1] and Dr Hughes[2] From these sources may also be gathered nearly all that is yet known respecting the various conditions under which the deposition of fibrine takes place, and the several forms which the deposits assume Into these general details I do not propose entering, my object being simply to consider the effects which the deposits may produce on the system at large It may, however, be premised that the forms of fibrinous concretions to which my observations chiefly apply, are, first, the masses usually described as Laennec's globular excrescences, and, secondly, the granular or warty growths adhering to the valves and presenting innumerable varieties from mere granules to large irregular fungous or cauliflower excrescences projecting into the cavities of the heart

Avoiding all discussion concerning the origin of these latter growths, I proceed at once to state that in whatever way they may originate, they are, when once formed, full of peril, and often remain so even long after the circumstances which gave rise to them have passed by If of large size and only loosely-adherent, as they often are, one or more masses of even considerable magnitude may at any time be detached from the

*Tr Roy Med-Chir Soc London 35 281-324 1852 Communicated by George Burrows, M D, F R S, Physician to St. Bartholomew's Hospital Received April 12, read May 25 1852

[1] Med Gaz vol xvi 1834-5

[2] Guy's Hosp Reports vol iv, 1839

valves and conveyed with the circulating blood until arrested within some arterial canal which may be completely plugged up by it, and thus the supply of blood to an important part be suddenly cut off, and serious, even fatal results ensue Or, the deposits on the valves may be detached in smaller masses, and pass on into arteries of much less size, or even into the capillaries, where, being arrested, they may cause congestion, followed by stagnation and coagulation of the blood, with all the subsequent changes which blood coagulated within the living body is liable to undergo In this way are probably induced many singular morbid appearances often observed in internal organs, and rarely well accounted for Again, the masses of fibrine may soften, break up, and discharge the finely granular material resulting from their disintegration, and this, mingling and circulating with the blood, may give rise to various disturbances indicative of a contaminated state of this fluid, producing symptoms very similar to those observed in phlebitis, typhus, and other analogous blood-diseases In one or more of these several ways, and probably in others not yet clearly recognised, fibrinous material detached from the valves, or any other part of the interior of the heart, may be the cause of serious secondary mischief in the body

It appears unnecessary to insist here on the possibility of any of the various forms of fibrinous deposit found within the heart being detached either spontaneously or by the mere force with which the current of blood passes over the surfaces on which they are placed For it is well known that after death a very gentle force, sometimes even the slightest touch, will loosen and dislodge both small granular particles and masses of considerable size from the valves and inner surface of the heart Not infrequently, indeed, lumps of old laminated fibrine of even considerable magnitude are found loose in the cavities of the heart, having probably dropped off before death, and sometimes a mass of this kind may be found some distance along the aorta or pulmonary artery

It is clear, then, that such fibrinous deposits may admit of being very readily detached, and it must be equally clear that once floating freely in the blood they are exposed to the almost certain consequence of being transmitted with this fluid, and stopped at the first vessel too narrow to allow of their transit

The parts of the vascular system within which these transmitted masses of fibrine may be found will of course depend, in great measure, upon whether they proceeded from the right or left side of the heart Thus if they have been detached from either the aortic or mitral valves, they will pass into the blood propelled by the left ventricle into the aorta and its subdivisions, and may be arrested in any of the systemic arteries or their ramifications in the various organs, especially those which, like the brain, spleen, and kidneys, receive large supplies of blood directly from the left side of the heart

If, on the other hand, the fibrinous masses are derived from the pulmonary or tricuspid valves, the pulmonary artery and its subdivisions within the lungs will necessarily become the primary if not the exclusive seat of their subsequent deposition A division of the subject being thus naturally formed, I propose to embody the remarks I am about to submit to the Society under two principal heads, considering—

1st The remote effects resulting from the separation of fibrinous or analogous deposits from the valves or interior of the left side of the heart, and

2d The corresponding effects produced by the detachment of like deposits from the valves or interior of the right side of the heart

PART I

ON THE EFFECTS WHICH MAY RESULT FROM THE SEPARATION OF FIBRINOUS DEPOSITS FROM THE VALVES OR INTERIOR OF THE LEFT SIDE OF THE HEART, AND THEIR CIRCULATION WITH THE SYSTEMIC BLOOD

In endeavouring to elucidate this part of the subject, I beg to draw attention, in the first place, to instances in which it seems probable that masses of considerable magnitude have been detached from the left side of the heart, and subsequently arrested in an arterial channel of notable size, secondly, to some of the effects which seem to ensue when smaller arterial vessels or capillaries are similarly blocked up, and, thirdly, to circumstances which make it probable that, not unfrequently, the introduction of particles of fibrine into the circulating blood gives rise to constitutional symptoms indicative of a poisoned state of this fluid

1 The first three cases which I shall offer are in many respects identical, for in each, death seemed to ensue from softening of the brain, consequent on obliteration of one of the main cerebral arteries by a mass of fibrinous material, apparently derived directly from warty growths on the left valves of the heart

Case I—Margaret Shaw, aet 34, a pale, weakly-looking woman, admitted into St Bartholomew's Hospital, under Dr Roupell, about the middle of July, 1850, on account of pains in her lower limbs, and general debility A loud systolic murmur was heard all over the cardiac region No material change ensued in her condition until August 7th, when, while sitting up in bed eating her dinner, she suddenly fell back as if fainting, vomited a little, and when attended to was found speechless, though not unconscious, and partially hemiplegic on the left side The hemiplegia increased, involving the left side of the face as well as the limbs, and gradually became complete in regard to motion, while sensation seemed to remain unimpaired She continued speechless and hemiplegic, but without loss of consciousness, for five days, when she quietly died

On examining the body, six hours after death, the skull and dura mater were found natural, but the small vessels of the pia mater were much congested, the congestion amounting, in some places, almost to ecchymoses. The right corpus striatum was softened to an extreme degree, being reduced to a complete pulp of a dirty greyish-white tint, and without any remains of its characteristic striated structure. The corresponding optic thalamus was healthy, but a condition of pale softening, similar to that affecting the corpus striatum, existed also to a considerable extent in the posterior lobe of the right cerebral hemisphere. The rest of the cerebral substance of this hemisphere was softer than natural, and appeared to contain less blood than ordinary. All other parts of the brain were healthy. The right middle cerebral artery just at its commencement was plugged up by a small nodule of firm, whitish, fibrinous-looking substance, which, although not adherent to the walls of the vessel, must have rendered its canal almost, if not quite, impervious. With the exception of a speck or two of yellow deposit in their coats, the rest of the vessels at the base of the brain were healthy and filled with dark blood.

The heart was enlarged; on its exterior were several broad white patches of old false membrane. The right cavities and left auricle contained recent separated coagula, the fibrine firm and whitish. The right valves were healthy, so also were the aortic, with the exception of slight increase of thickness. The mitral valve was much diseased, the auricular surface of its large cusp being beset with large warty excrescences of adherent blood-stained fibrine. There were a few scattered deposits in the coats of the aorta. The right common iliac artery, about an inch above the origin of its internal branch, was blocked up by a firm, pale, laminated coagulum, which extended into the internal iliac, and for about a quarter of an inch down the external iliac, where it terminated rather abruptly. The lower portion of the coagulum was colourless, and softer and more crumbling than the upper, which was also more blood-stained and laminated. There was no adhesion of the coagulum to the walls of the vessels. No similar clot existed in the iliac vessels on the opposite side. The pleurae were adherent in places, the lungs oedematous, and in places solidified by compact greyish-white masses, such as might result from uncured pneumonia. The pulmonary vessels were free from old coagula.

The liver and intestinal canal were healthy. The spleen was enlarged, pale, and soft. One large portion, about a fourth of the organ, was converted into a mass of firm, yellowish-white, cheesy substance. The kidneys were pale, rough, and granular. Within the cortex of the right were several large masses of yellow deposit, surrounded by patches of redness. The portions of medullary structure passing to these deposits were compact, dryish, and yellow.

In the case just narrated death evidently resulted from softening of a large portion of the right side of the brain, and the cause of this softening appeared to be an imperfect supply of blood, consequent on the middle cerebral artery of the same side being obstructed by a plug of fibrine within its canal. I am not aware that there has yet been recorded a case in which fatal softening of the brain resulted from a cause like this, therefore in itself this case is one of value. That the existence of the fibrinous coagulum within the cerebral artery was the real cause of the changes in the brain, can, I think, scarcely admit of question. The sufficiency of such an obstruction to produce the effects ascribed to it is fully established by the many instances in which disturbance, or complete arrest of function in a part, with subsequent atrophy or disorganisation of its tissue, results from any circumstance which materially impedes or entirely cuts off its supply of blood.

PART II

ON THE EFFECTS WHICH MAY RESULT FROM THE DETACHMENT OF FIBRINOUS DEPOSITS FROM THE RIGHT VALVES OF THE HEART

If, from what has been stated, it be assumed as probable that deposits of fibrin occurring on the valves of the left side of the heart, may, by being detached, be productive of serious affections of remote organs, it may be inferred also that similar deposits occurring on the right valves may induce corresponding secondary affections of the lungs. And there seems to be sufficient evidence for believing that such is really the case. For it may, I think, be clearly shown, that most of the fibrinous or other similar secondary deposits in the lungs, also many of the old coagula found in the pulmonary artery or its branches, and possibly some forms of pulmonary apoplexy, are closely connected with it, if not actually dependent upon, fibrinous deposits on the valves, or interior of the right side of the heart, or materials transmitted through the heart by venous blood. It is of course conceivable that when the deposits on the right valves consist of large warty masses, as they occasionally do, portions of considerable size may be detached, and transmitted along the pulmonary artery, and so plug up one of the large branches of this vessel, just as similar masses detched from the left valves may be arrested in one of the main systemic arteries, but I have not yet met with a decided instance of such an occurrence. Probably the more usual manner in which the separation of fibrinous masses from the right valves leads to the formation of coagula in the pulmonary artery, is by the transmission of small particles to the minuter divisions of the artery, or to the capillary plexus, arrested at which they induce stagnation of the blood in those branches

of the artery distributed to the seats of obstruction. Such a result is almost necessarily consequent on the peculiar mode of distribution of the branches of the pulmonary artery, which pass to their destination without anastomosis. In a paper on the formation of coagula in the pulmonary artery, published in the Transactions of this Society, Mr. Paget has clearly shown the influence which certain obstructions in the pulmonary capillaries, such as oedema, chronic pneumonia, and pulmonary apoplexy, sometimes exercise in inducing coagulation of blood in the arteries supplying the obstructed parts. And I have likewise noticed a similar influence apparently resulting from other circumstances, such as extensive old tubercular disease, and extreme compression of the lung by false membrane on the pleura, which have obliterated large portions of the pulmonary tissue. Mr. Paget also narrates instances in which particles of cancerous matter brought from remote organs to the right side of the heart, and thence transmitted to the lungs, became arrested in the pulmonary capillaries, and so induced stagnation and subsequent changes of the blood, in branches of the pulmonary artery. Cases like these, of which I have seen several examples, seem to leave no doubt that a like coagulation of blood in the pulmonary arteries may result from obstruction caused by the arrest of particles of fibrine detached from the right valves of the heart, and transmitted to the pulmonary capillaries. An instance of this is furnished by one of the cases already narrated (Case III), in which, together with large, nodular, and warty masses attached to the tricuspid valve, nearly every branch of both divisions of the pulmonary artery were blocked up by old fibrinous coagula.

Another equally striking illustration is afforded by a specimen in the museum of St. Bartholomew's Hospital, in which, with extreme disease of the pulmonary valves, accompanied with the deposition of thick irregular layers of soft fibrine on each of them, there were old coagula filling many of the branches of the pulmonary artery. In this case there were also several large, solid, fibrinous masses in the substance of the lung, and it seems reasonable to believe that these, as well as the coagula in the pulmonary artery, had their origin in the deposits of fibrine on the pulmonary valves, portions of which were probably detached, arrested in the capillary plexus of the lungs, and so caused the fibrinous masses in the pulmonary tissue, and the consequent coagulation of blood in the arterial branches distributed to these parts.

The fibrinous masses in the lungs which the specimen just mentioned presents, appear not unlike portions of old pulmonary apoplexy, from which most of the colouring matter of the extravasated blood has been removed, and it is not improbable that many similar masses in other cases may have originated in a like cause, and not in haemorrhage into the pulmonary tissue. Such masses, indeed, represent one of the appearances described as capillary phlebitis of the lungs, or, in other words, one

stage in the transformation undergone by blood stagnant and coagulated in the pulmonary capillaries. This blood passes through the same changes in the lungs that it undergoes when similarly situated in other organs, and the various examples of these changes are not infrequently met with in the lungs. Thus, in Case III were found various gradations, from firm compact coagula, through soft, brownish, disorganised blood, to collections of yellowish, puriform material, which in places formed ordinary abscesses. Masses of such large size and with such obvious characters as these, are of course readily recognised. Yet not infrequently, deposits of a similar nature exist in the lungs, though of such extreme minuteness as to elude detection, unless specially sought for. These consist of small, slightly elevated, red dots, with a pale-yellow or buff-coloured centre, scattered, sometimes thickly, over the surface and within the interior of the lung. They are exactly identical with that spotted form of capillary phlebitis already mentioned as often occurring in systemic organs and in various tissues, either combined with other forms or alone. When met with in the lungs I have hitherto invariably found it either as the result of some morbid material in the venous blood, or in direct connection with affection of the right valves of the heart, such affection, namely, as is attended with the deposition of fibrinous granules on the surface of the valves. To quote but one instance out of several of the kind, I would mention the case of a girl under the care of Dr Hue, early in the year 1851. This patient died suddenly, after suffering for some months with symptoms of extreme disease of the heart. Besides general enlargement of the heart, and narrowing of the mitral orifice, the free border of the tricuspid valve was studded with small, pale, fibrinous granules, a few of which existed also on the pulmonary valves. At first sight the lungs appeared healthy, but, on closer inspection, they were found freckled throughout with small, dark-red spots, like minute ecchymoses, in the interior of several of which was a distinct buff-coloured speck. The view which may not unreasonably be taken of these spots is, that they consisted of congested capillaries, in which minute fragments of fibrine, transmitted from the right valves of the heart had been arrested, the appearances, indeed being just such as resulted from the injection of softened meat into the blood in one of M. Gaspard's experiments.

Under whatever form these various deposits are met with in the lungs, I believe that careful examination will show them to be almost invariably associated either with the presence of fibrinous growths on the right valves of the heart, or with some other condition leading to the existence of particles of fibrine or other foreign matter in the blood transmitted to the lungs. Of these other conditions the most important seem to be the disintegration of old masses of fibrine situated within the right cavities of the heart, and a like disintegration of old coagula in some part of the

venous system, and its subsequent mixture with the venous blood It appears to be quite usual for the old colourless or pale-reddish clots found in the right cavities of the heart, especially in the appendix of the auricle, to soften in the centre, and be converted into a dirty reddish-brown or fawn-coloured material Sometimes the softening extends through the whole substance of the mass, with the exception of a thin layer at the circumference which forms a kind of cyst or bag within which the softened material is contained Sometimes too this cyst bursts and discharges its contents, leaving nothing but the outer shell attached to the interior of the heart The softened material thus left loose and mingled with the blood will doubtless contaminate it almost as effectually as the direct introduction of a similar material by injection into a vein would do And it is easy to imagine that the solid particles of fibrine may be arrested at the capillaries of the lungs, and produce the various forms, especially perhaps the spotted variety, of deposit to which allusion has been made Old coagula in the veins too, under whatever circumstances they may have originated, appear almost equally liable to undergo softening, and to break up and mingle their disintegrated particles with the venous current along which they may pass to the lungs, and produce effects similar to those consequent on the transmission of like material from the cavities of the heart

Such are some of the principal effects which the transference of fragments of fibrine from the right side of the heart appear capable of producing in the lungs Much more might be said on the subject, but the length to which this communication has already extended precludes any further remarks at the present time I would only add the suggestion that possibly the peculiar form of the pneumonia sometimes observed in rheumatic fever may, in some way, have its explanation in the transmission of fibrinous particles from the right valves of the heart to the lungs The almost invariable existence of disease of the pulmonary or tricuspid valves in the fatal cases of rheumatic pneumonia I have examined after death strongly favours the opinion that there is some close relation between this peculiar inflammation of the lungs and the fibrinous deposits on the right valves of the heart

In conclusion, let me briefly recapitulate the principal points I have endeavoured to establish to the satisfaction of the Society They are, 1st, the general fact that fibrinous concretions on the valves or the interior of the heart admit of being readily detached during life, and mingled with the circulating blood, 2dly, that if detached and transmitted in large masses, they may suddenly block up a large artery, and so cut off the supply of blood to an important part, if in smaller masses, they may be arrested in vessels of much less size, and give rise to various morbid appearances in internal organs, while, under other circumstances, the particles mingled with the blood may be extremely minute, possibly the

debris of softened fibrine, yet in sufficient quantity and with sufficient power to produce a poisoned state of the circulating fluid, as manifested in the production of typhoid or phlebitic symptoms 3dly, that the effects produced and the organs affected will be in great measure determined by the side of the heart from which the fibrinous masses have been detached, for, if the right valves have furnished the source of the fibrine, the lungs will bear the brunt of the secondary mischief, displaying it in coagula in the pulmonary arteries, and various forms of deposit in the pulmonary tissue but if, as is far more commonly the case, the left valves are affected, the mischief is more widely spread, and may fall on any systemic part, but especially on those organs which, such as the brain, spleen, and kidneys, are largely and directly supplied with blood from the left side of the heart

1854

WILLIAM STOKES

DESCRIPTION OF THAT PERIODIC FORM OF BREATHING LATER TO BE KNOWN AS CHEYNE-STOKES RESPIRATION

FATTY DEGENERATION OF THE HEART*

By

WILLIAM STOKES†

GENERAL DIAGNOSIS OF THE DISEASE

IF IT be inquired how far we have gone, since the time of Laennec, in establishing the diagnosis of this affection, it will appear that as yet but little has been done. Laennec declared that he knew of no means by which the diagnosis of fatty degeneration of the heart could be made, and Dr. Ormerod, writing in 1849, observed, that "the most extreme cases detailed may show that the diagnosis on general or physical grounds is almost impossible." "We cannot," he says in another place, "predict with certainty in any case that we shall find this lesion after death, but it is difficult for any pathological observer not to be led to suspect the existence of a disease in the repetition of the same circumstances under which he has seen it occur previously."

The diagnosis of this condition is not only possible but often free from difficulty, at least where the disease is confirmed. On the other hand, minor degrees of the affection are to be determined less by direct signs than by some general characters.

The diagnosis turns upon three points:—

1. The existence of physical signs and symptoms of diminished force of the heart.

2. The occurrence of certain symptoms, principally referrible to the brain, which indicate either anaemia on the arterial, or congestion on the venous side, of the cerebral circulation.

3. Symptoms referrible to the respiratory function, which appear to arise from deficient power in the right ventricle.

It is still to be determined how far we can distinguish during life the cases of weakened, and dilated hearts, such as have been already described, from those of fatty degeneration. Microscopical anatomy shows that in many of the former class there is more or less of the adipose deposit. And it is plain that to the practical physician there is a relation

*Stokes, William. *The Diseases of the Heart and the Aorta*, Dublin 1854, Hodges and Smith, pp. 320-327. We reprint from Medical Classics 3: 739-746, 1939.—F. A. W. 1940.

†For an account of Stokes's life see pp. 459-461. Stokes's paper 'Observations on Some Cases of Permanently Slow Pulse,' is reprinted on pp. 462-469.

between the diseases, for similar exciting causes concur in their production, and in both the effect of the disease is traceable to the same vital condition, namely, debility of the heart

In its higher degrees of development this affection is most frequently met with in persons who have passed the prime of life, but minor shades of it occur in young patients, especially where there is a complication with other visceral diseases, as, for example, pulmonary tubercle On the other hand, some of the most remarkable instances are found in very old and long bedridden subjects, and it is observed that in such cases the alteration is not confined to the heart, but extends also to the voluntary muscles, and even to the skeleton, producing atrophy and fragility of the bones, with a great deposit of oily matter in the cavities and cancelli of the osseous tissue * Though varying and apparently opposite, its exciting causes are generally reducible to those which would induce a depraved haematosis The over-fed and luxurious, on the one hand, and the victim of want, on the other, are liable to the disease

Although complication with various local diseases, or with a special morbid state such as gout, is not uncommon, yet judging from the good state of the general health, and the absence of lesion in the digestive, respiratory, and nervous systems after death, we must admit that the fatty heart may be, in a large number of cases, practically considered as a local affection

It is probable that in these uncomplicated examples, the disease attains its greatest development, and exhibits the most characteristic symptoms

The symptoms may be divided into those referrible to the nervous, respiratory, and circulating systems

Of the nervous symptoms, the most important are the attacks of apoplexy, or pseudo-apoplexy, to which these patients are so liable This affection differs from ordinary sanguineous apoplexy in three particulars, namely, the frequent repetition of the seizures, the rarity of consequent paralysis, and the fact that there is not only danger from an antiphlogistic treatment, but benefit, both remedial and preventive, from the use of stimulants

In some cases the character of these attacks approaches to that of syncope, and it is difficult to say how much of the affection is produced by the want of arterial, or the stasis of venous blood In the earlier periods of the case the attack is more of syncope, in the later it becomes apoplectic The attacks may occur without warning, and the first seizure be fatal This, however, is rare In most cases there are numerous seizures at irregular intervals, and in some, sensations referrible to the epigastrium and head, having a resemblance to the epileptic aura, give notice to the patient that he is about to be attacked In some there is a

*Of this condition numerous specimens may be seen in the Museum of the Richmond Hospital

momentary unsteadiness in walking, and in others a tendency to faint, which may be dissipated by any ordinary stimulus, while in the more decided cases the patient becomes suddenly comatose, a condition which may be preceded by loss of memory and a lethargic state. I have at present under my care a patient whose earlier attacks were syncopal, they are now apoplectic, and come on during sleep, each one being preceded by a slight convulsion. On recovery, and after all the comatose symptoms have passed away, he remains for half-an-hour unable to recognize his most intimate friends and relations, even his wife he has mistaken for his mother. This patient is 63 years of age. This latter symptom has been observed in a case of weak heart which lately occurred in Dublin, the patient frequently failing to recognize friends who had been his intimates for half a century. The duration of the attack is generally short, paralysis is rare, and when it occurs does not seem referrible to any anatomical lesion of the brain. The question as to whether these singular attacks are dependent upon deficient arterial supply, or rather upon venous congestion, is a difficult one, but it does not involve any important point of practice. It is true, that whatever arrests the action of the heart will retard the flow of blood in the veins of the head, but it could not cause a state of hyperaemia. The opinion that the apoplectic seizures are owing to deficient arterial supply seems the most tenable. The suddenness of the attack, and, in many instances, the rapidity of the recovery, are in favour of this view. I have noticed one case in which, on the occurrence of the premonitory symptoms, the patient, by hanging his head so that it rested on the floor, used to save himself from an attack. A case lately occurred to me of an aneurism of the aorta, in which three successive ruptures of the sac took place, with intervals of several days. Each rush of blood was attended with the best-marked syncopal coma and convulsions. Finally, dissection does not show any extra-ordinary congestion of the brain, and we learn from auscultation that the action of the heart is feeble.

This view of the cause of the attacks appears to be still further corroborated by the occurrence of symptoms of a similar nature in the case of a dilated mitral opening by Dr. Fleming, which has been already given. Here the ventricle was hypertrophied to a great degree, but the patient suffered from regurgitation into the left auricle.

We can, therefore, only adopt in part the plan of treatment suggested by the late Mr. Carmichael, which was to relieve the vessels of the head by venesection, while at the same time stimulants should be used to excite the action of the left ventricle.

Symptoms referrible to the respiratory function.—There is no evidence that the existence of this disease, even in an aggravated form, is an exciting cause of any organic affection of the lung. On the other hand, the

researches of Ormerod, Quain, and others, have demonstrated the frequent combination of fatty heart with pulmonary disease, but in such cases we may hold that the conditions of the lung and heart have little, if any mutual relation, they are rather to be considered as the secondary accidents of a general morbid state

But there is a symptom which appears to belong to a weakened state of the heart, and which, therefore may be looked for in many cases of the fatty degeneration I have never seen it except in examples of that disease The symptom in question was observed by Dr Cheyne although he did not connect it with the special lesion of the heart It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnoea is established In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations This symptom, as occurring in its highest degree, I have only seen during a few weeks previous to the death of the patient I do not know any more remarkable or characteristic phenomena, than those presented in this condition, whether we view the long-continued cessation of breathing, yet without any suffering on the part of the patient, or the maximum point of the series of inspirations, when the head is thrown back, the shoulders raised and every muscle of inspiration thrown into the most violent action, yet all this without râle or any sign of mechanical obstruction The vesicular murmur becomes gradually louder, and at the height of the paroxysm is intensely puerile

The decline in the length and force of the respirations is as regular and remarkable as their progressive increase The inspirations become each one less deep than the preceding, until they are all but imperceptible, and then the state of apparent apnoea occurs This is at last broken by the faintest possible inspiration, the next effort is a little stronger, until, so to speak, the paroxysm of breathing is at its height, again to subside by a descending scale

In other cases we see the symptom of sighing to occur in a different manner at irregular intervals the patient draws a single deep sigh, especially when he suffers from fatigue, want of food, or of his ordinary stimulants This is the commonest form of the affection* In one case it was always most evident when the patient was lying down

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*The sighing respiration may be observed in persons who are labouring under certain forms of gastric or hepatic derangement, and is occasionally a symptom of undeveloped gout It disappears under appropriate treatment, and probably indicates a temporary weakness of the heart I lately saw a case of long-continued sighing, in which it had apparently arised from depression and anxiety of mind, but had, as it were, become a habit The patient was a lady of very nervous disposition A feeble murmur attended the first sound of the heart In this case there was probably no organic lesion, for the symptom had long existed, and there were no signs of progressive disease

(Footnote continued on page 488)

The phenomena of circulation are next to be considered We are in want of a sufficient number of observations to enable us to declare whether in the earlier periods there is any marked character of pulse as to strength, frequency or regularity Many of the recorded cases of the minor stages of the disease are deficient in accurate observations of the pulse, but it may be held that no special character of pulse has been established In some the pulse has been weak, rapid, and irregular, in others it does not seem to have differed materially from that of health * But in confirmed cases we may meet with three important characters of pulse —

1 The pulse somewhat accelerated, but occasionally intermitting, its strength may be but little altered

2 The extremely weak, rapid, irregular, and tingling pulse (pulsus formicans)

3 The permanently slow pulse, the rate of which varies from 50 to 30 in the minute, or even less

It is probable, that in the third class of cases, or those with a permanently slow, though distinct and regular pulse, the disease has either advanced to a great degree, or has at all events affected the different portions of the heart equably, and that we may attribute the weak and irregular pulse to conditions of the heart in which only certain portions of the organ have degenerated, or where there is a great difference between the right and left sides of the organ It is further probable that the heart may be in two very different conditions previous to the commencement of the fatty change, and that in the case with irregular pulse, a merely weakened and perhaps dilated condition has preceded the deposit of fat globules in the muscular fibre, while in the third class the change

(Footnote continued from page 487)

Sufficient attention has not as yet been directed to this character of respiration It is when confirmed almost pathognomonic of a weak and, in all probability, a fatty heart, but whether it is to be taken as indicative of the predominance of the fatty change on the right side of the heart is still an open question Laennec has described a form of asthma with puerile respiration and he attributes the disease and the signs to some special modification of the nervous influence He observes that he has never met with it except in persons affected with mucous catarrh and holds that dyspnoea arising from the mere increase of the natural want of the system for respiration, can never amount to asthma without the catarrhal complication But he further speaks of adults and old persons who have puerile respiration without catarrh and who, though they are not, *properly speaking, asthmatic are short-breathed, and liable to dyspnoea on the slightest* exercise

It is possible that in some of these cases at least, the heart may be in an incipient stage of fatty degeneration I have observed the symptom in a gentleman of about 70 years of age, who has many symptoms of a weak heart The action of that organ is regular but the *impulse is extremely feeble and the pulse compressible* The sounds especially the first are very indistinct there are no bronchial rales but well-marked puerility of respiration exists over every portion of the thorax He principally complains of dyspnoea on exercise or any mental agitation and the symptoms have only become prominent within the last eighteen months So far as the permanent condition of the respiration is concerned this case answers perfectly to Laennec s description of dyspnoea with puerile respiration See Dr Forbes s translation of the work of Laennec—Article, Asthma with Puerile Respiration

*This circumstance is worthy of consideration in connection with that which I have recorded as occurring in cases of the softening of the heart in typhus, in many of which the pulse is quite a fallacious guide in determining the strength of the left ventricle

has occurred without previous alteration in the structure or mode of action of the heart Some of the cases observed in persons who have been long bedridden, and who have died from rupture of the left ventricle, are of this description Additional observations, however, are necessary to elucidate this subject

If we inquire whether irregularity of pulse is indicative of valvular disease in this affection we must consider that the symptom may be met with in cases of weak dilated hearts, without valvular disease, and therefore, that we might expect it in fatty degeneration On the other hand, the occurrence of cases with a perfectly regular though slow pulse is a remarkable fact In well-marked cases where irregularity, rapidity, and smallness of pulse exist, we ought not, even though there be no valvular murmur to declare too strongly against the existence of valvular obstruction, bearing in mind, first, that the very weakness of the heart may prevent the appearance of murmur, and next that valvular disease is not infrequent combination with fatty heart In most of the cases which I have seen, this valvular affection was at the aortic orifice, and the pulse was slow and regular

1861

PAUL LOUIS DUROZIEZ

DESCRIPTION OF THE DOUBLE INTERMITTENT MURMUR OVER THE FEMORAL ARTERIES IN AORTIC INSUFFICIENCY, LATER TO BE KNOWN AS DUROZIEZ'S SIGN

PAUL LOUIS DUROZIEZ

(1826-1897)

"The Physician sees patients and not diseases"

—Duroziez, in Preface to
Traité Clinique des Maladies du Coeur, 1891

PAUL LOUIS DUROZIEZ was born in Paris on January 8, 1826 He was a student at L'Institution Favart, a Parisian grammar school Later he became a student at the Lycée Charlemagne, where he excelled in Latin, Greek, and English, and in 1844 was graduated with the degree, Bachelor of Letters and Sciences

Duroziez then began the study of medicine at the Faculté de Médecine of Paris and in the Paris hospitals He studied under Alfred A L M Velpeau (1795-1867) in 1845 and 1846 Later, in 1848, he became an extern to Dr Blache at the Hôpital des Enfants Malades In 1849 and 1850 he was an extern under Professor Jean Baptiste Bouillaud (1796-1881) at La Charité in Paris In 1850 he was awarded the Corvisart Prize for a clinical study of the subject, "Therapeutic Properties and Physiologic Action of Digitalis," the subject having been designated by the Faculty of Medicine On July 21, 1853, he received the degree of Doctor of Medicine, his thesis was entitled "Clinique de la Charité, Service de M le Professeur Bouillaud, Sémestre d'Hiver 1850-1851"

In 1856 he was named chief of a clinic at La Charité in Bouillaud's service, where he remained until 1858

His marriage to Mademoiselle Rohan, who was of Bourbon lineage and whose grandparents had been guillotined during the French Revolution, took place on June 25, 1861 Four children resulted from this marriage The eldest, a boy, died at the age of eight from scurvy which developed during the siege of Paris in 1870 The other three children were daughters, each of whom received an excellent education The year of his marriage (1861) Duroziez published his important paper on "The Double Intermittent Murmur over the Femoral Arteries as a Sign of Aortic Insufficiency" This murmur later became known as "Duroziez's sign" We are including an abstract, in translation, of this paper in our Cardiac Classics Duroziez also elucidated in 1861 the pure type of mitral stenosis, setting forth its clinical character This later became known as "Duroziez's disease"[1]

In 1867, Duroziez was appointed physician of the Bureau of Welfare of the First Ward of Paris During the Franco-Prussian War, in 1870, he served as ambulance physician and surgeon major of the Fourteenth Infantry Battalion For his outstanding work with patients during the smallpox epidemic he was awarded a silver medal from the Minister of the Interior

In 1879, he was appointed a member of the Commission of Public Hygiene and Health of the First Ward of Paris, and in 1889 he was appointed medical inspector of schools of the same ward In 1891, he was awarded the Itard Prize of the Academy of Medicine for his work, "Traité Clinique des Maladies du Coeur" In 1891, also, L'Institut de France awarded him the Montyon Prize for the same work

[1]Duroziez P Du rétrécissement mitral pur Arch gén de méd **140** 32-54 1877

At the age of seventy, on January 4, 1895, Duroziez was named a chevalier of the Legion of Honor. The honor came when he had ceased to care much about it, and to the patient who brought him the news he exclaimed: "What a good thing for my wife!"

For thirty years Duroziez occupied an important place in the Société de Médecine, before which body he made many original contributions which were subsequently published in the "Communications" of that society. He was its president in 1882.

Early in 1897, Duroziez contracted pneumonia, to which he succumbed at noon on January 16, 1897.

Duroziez always professed a profound and genuine interest in the diseases of the heart. "As long as my own heart beats," he said to a contemporary, "I shall continue to auscultate the one of others." He considered that organ as a separate being endowed with a male half, the left ventricle, and a female half, the right ventricle. The former, he thought, was calm, regular and stable, the latter, he considered, was nervous, impressionable, and often disordered. In his original work on the duality of the heart[2] Duroziez referred to the four cardiac cavities, comparing them *to four horses fastened to the same chariot*. He pointed out that such an arrangement permitted an easy break in equilibrium with resulting badly combined movements.

Duroziez once remarked, "In the heart, bruits are too full of detail, too brief. The cavities do not always have the same relationship. One does not know where one is." In spite of this remark, Helfenbein wrote that Dr. Jullien said of Duroziez, "Bruits were to him sweet music, he listened to them in the chest, surprised them in the back, pursued them into the neck and even into the thigh."

Duroziez also introduced the onomatopoetic Fout-tata-Rou which he used to describe the various signs in the heart heard in mitral stenosis. Among his many observations, mention should be made of his description of the sequelae of pure mitral stenosis, such as embolism, aphasia, and right hemiplegia. He also noted the predominance of this disease among women.

[2]Duroziez, P. De la dualité du coeur, Bull. Soc. de méd. de Paris 27: 39-42, 1893.

THE DOUBLE INTERMITTENT MURMUR OVER THE FEMORAL ARTERIES AS A SIGN OF AORTIC INSUFFICIENCY

By

DR P DUROZIEZ

Former chief of the Clinic of the Faculty at the Charite Hospital (Service of Professor Bouillaud)

The femoral arteries, which are subjected to auscultation less frequently than the carotids, offer very valuable information, they are especially unique in their behavior, they are readily compressed and in this respect offer the same advantages as the radial arteries they are larger than the carotid arteries and have the advantage of being more distant from the heart

The femoral artery merits careful study

On compression of the femoral artery, a shock or thrill is felt and auscultation reveals a sound, similar to the sound of *toc* or a sound of unique blowing character, a simple intermittent blowing murmur The entire femoral artery is capable of giving rise to this blowing murmur The character of the murmur varies with changes in the blood, the size of the artery, the condition of the vessel wall, and the contractile force of the heart After compressing the artery for some time and gradually releasing the pressure in a subject with chlorosis, a continuous humming murmur will appear, at times a continuous, humming sound is audible, at other times a double murmur is audible

The so-called intermittent double murmur which occurs in certain cases, is a different murmur and our study will concern it

The intermittent double murmur over the femoral arteries was described in aortic insufficiency, but no one, I believe, has given it the significance that it deserves Everyone has mentioned the murmur occurring in arterial diastole (*souffle de la diastole arterielle*) which quite frequently occurs without compression of the artery, but very few authors mentioned the murmur occurring during systole Very frequently it does not appear of its own accord, but must be produced and sought for The

*Du double souffle intermittent crural comme signe de l'insuffissance aortique Arch gén de méd Paris 107 417-443 588-605 1861 Translated by Erich Hausner M D Amsterdam New York

first murmur results from the powerful contraction of the ventricle, but as the second murmur is produced by the systole of the arteries in the legs, a less powerful force, its production must be facilitated by compression of the artery.

In cases of uncomplicated aortic insufficiency, wherein the heart beats vigorously and the arteries pulsate and react forcefully, the double murmur is audible; when, contrarily, aortic insufficiency is complicated by a considerable degree of aortic or mitral stenosis, a not uncommon occurrence, the arteries are moderately distended with blood and thus the second murmur is difficult to hear. It must be carefully sought and even then it will not appear regularly; it will not be detected when weak pulsations are present. It appears or disappears in relationship to increased or decreased action of the heart. At times it can be heard over both femorals, at other times only over one; briefly, distention and recoil, adequate systole of the arteries, are required for its presence; a careful examination is indispensable.

The double murmur can be produced in two ways, by means of the stethoscope or by means of the hand. With the stethoscope pressure is exerted to completely compress the artery; at a certain moment the double murmur will appear; only when the second murmur can be readily produced is it possible to place the stethoscope on the artery without pressure and then gradually slight pressure can be exerted with the hand above and below the stethoscope. Pressure above will produce the first murmur, while pressure below will produce the second murmur; it is evident that the second murmur is produced by the arteries of the legs, which propel the blood backwards and in some manner empty the capillaries.

The double intermittent murmur is of interest not only from the standpoint of diagnosis. The reflux of blood explains some of the symptoms occurring in aortic insufficiency and explains the sudden death which is occasionally observed.

A great disturbance occurs in the circulation; the blood no longer circulates evenly, so to speak, but comes and goes into the arterial system and stagnates in the veins, which continuously try to empty themselves. In the presence of aortic insufficiency, the heart during its powerful diastole, aspirates the blood from the lungs through the pulmonary veins at the same time that it receives the blood from the capillaries; the right ventricle and the lungs are emptied of blood. The blood supply is poor, the patients are pale, die from anemia and syncope. They do not tolerate venesection well.

What a difference occurs with mitral stenosis! Here, on the contrary, the blood is stagnant, forced into the veins, into the right side of the heart and the lungs; the patients die from apoplexy and suffocation; venesection gives relief.

These are two conditions, in opposition to each other, and one may be considered as being beneficial to the other

Auscultation is an important issue in this connection We have distinguished by our observations the auscultatory phenomena and particularly those concerning the femoral arteries

1 In all heart cases wherein the double intermittent murmur was audible over the femoral arteries, aortic insufficiency was found at autopsy

Conclusions

1 The double intermittent murmur audible over the femoral arteries, described by many authors in aortic insufficiency, has to my knowledge never been given as a constant sign of this lesion

2 Most commonly it is not present and it is necessary to produce it by compression

3 In aortic insufficiency blood is first propelled from the left ventricle into the extremities, and, being repulsed by the peripheral arteries and drawn back by the left ventricle, flows from the extremities towards the heart

4 The finger, compressing the artery about two centimeters above the stethoscope, produces the first murmur, two centimeters below, the second murmur

5 The secondary murmurs which can be produced by lesions of the pericardium, by mitral stenosis, tricuspid stenosis, by pulmonary insufficiency, can be differentiated from the murmur of aortic insufficiency with the help of the double murmur over the femoral arteries, which exists only in the latter condition

6 If aortic insufficiency is complicated by one or more of the lesions mentioned, and if the diagnosis is rendered difficult by these complications, the phenomena in the femoral arteries will help or even establish the diagnosis

7 The femoral phenomena less clearly differentiate aortic valvular lesions and lesions of the aorta The double murmur may appear in certain aneurysms without insufficiency being demonstrable after death

8 The temporary insufficiency can be demonstrated by the evanescent intermittent double murmur

9 A continuous murmur can originate in the arteries, this, however, is never audible in aortic insufficiency with its constant intermittent double murmur over the femoral arteries

10 The double intermittent femoral murmur occurs in typhoid fever, chlorosis, lead intoxication, but only temporarily, it is soon replaced by continuous murmurs

1862

AUSTIN FLINT

DESCRIPTION OF THE MURMUR LATER TO BE KNOWN AS THE AUSTIN FLINT MURMUR

AUSTIN FLINT

(Courtesy Charles C Thomas)

AUSTIN FLINT

(1812-1886)

"The Watson of America"

—*The Lancet* (London)

FOUR years before Laennec happened upon the discovery of mediate auscultation, Austin Flint was born on October 20 at Petersham in Massachusetts He was the son of Dr Joseph Henshaw Flint, a well-known physician and surgeon of Northampton, Massachusetts His grandfather, Dr Austin Flint of Leicester, served as a surgeon in the war of the American revolution, and his great-grandfather, Dr Edward Flint, was a noted practitioner from Shrewsbury, Massachusetts

Reared in such an impressive professional tradition, it was not surprising that young Austin Flint should join the ranks of American physicians Nor did this line of physicians halt with the subject of our memoir, for his son, Austin, Junior, was a physician So, also, was Austin Junior's son, who at this writing is professor emeritus of obstetrics in the New York University College of Medicine Those who are proud of the achievements of American medicine feel a real debt of gratitude to the Flints, who have greatly enriched American medicine, and especially to Austin Flint, Senior, one of the most important American pioneers, whose achievements have been linked with those of Skoda, Stokes, and Walshe

Austin Flint, Senior, was a student at Amherst and Harvard Colleges He studied medicine at the Harvard Medical School and was graduated from that institution in 1833 Flint was fortunate in having among his teachers Jacob Bigelow (1787-1879) and James Jackson (1777-1868) Bigelow was professor of materia medica at Harvard, author of the three-volume "American Medical Botany" (1818-1820), and one of the greatest of American botanists Jackson had studied medicine in St Thomas' Hospital (London) where he had been a student of Astley Cooper He became professor of medicine at Harvard and during Flint's student days was much interested in the subject of physical exploration Flint said of him "He never failed to carry the stethoscope during his hospital visits, and the signs of cardiac and pulmonary disease entered largely into his clinical instructions" Oliver Wendell Holmes said Jackson was

"Thoughtful in youth, but not austere in age,
Calm but not cold, and cheerful though a sage"

For the first three years after his graduation, Flint practiced medicine at both Northampton and Boston, Massachusetts He then moved to Buffalo, New York In 1844, he accepted the professorship of the theory and practice of medicine at Rush Medical College, Chicago, retaining his residence in Buffalo He held his post at Rush for a year, at the end of which he resigned to establish the "Buffalo Medical and Surgical Journal" He was the editor of this publication for ten years

In 1847, Flint, with Dr James P White and Dr Frank H Hamilton, founded the Buffalo Medical College, which is now the School of Medicine of the University of Buffalo At Buffalo Medical College Flint taught the theory and practice of Medi-

cine, as well as clinical medicine, until 1852, when he resigned to become professor of the theory and practice of medicine at the University of Louisville.

Flint served four years at Louisville after which (1856) he returned to Buffalo where he accepted the chair of pathology and clinical medicine in the school he had helped to found. Flint spent the winter seasons from 1858 to 1861 at New Orleans, Louisiana, where he filled the professorship of clinical medicine at the New Orleans School of Medicine and was attending physician to the Charity Hospital.

In 1859 Austin Flint accepted two faculty appointments in New York City. He became professor of the principles and practice of medicine and clinical medicine and visiting physician at Bellevue Hospital Medical College. He also accepted the professorship of pathology and practical medicine at the Long Island College Hospital. He resigned from the latter position in 1868.

Flint was a member of many medical and scientific societies in America and Europe. He was elected to the presidency of the New York Academy of Medicine for the term, 1873-1874. In 1883 he was elected president of the American Medical Association, an office which he had not sought and which he did not desire. He had been made chairman of the Section on Practical Medicine in 1850, and in fact, had helped to found the Association in 1848.

At the meeting of the International Medical Congress in London in 1881, Dr. Flint read a paper on "The Analytical Study of Auscultation and Percussion with Reference to the Distinctive Characteristics of the Pulmonary Signs." This paper was received with such acclaim that Flint was asked to serve as chairman of a newly formed committee which was to report on a "Uniform Nomenclature of Auscultatory Sounds in the Diagnosis of Diseases of the Chest." The committee's report was made at Copenhagen in 1884. Flint's suggestion, made with Dr. Samuel D. Gross, led to the decision that the International Medical Congress should convene in 1887 in the United States. Flint was to have succeeded Samuel D. Gross as president of this body, but Gross died in 1884, on the very day on which Flint, as president of the American Medical Association, asked that the Congress meet in America. Flint was elected president of the Congress, but died in 1886, before it met.

Austin Flint was a prolific writer on all phases of medicine. It is outside the scope of this brief sketch to list all his contributions to the literature, but mention may be made of his outstanding contributions to physical diagnosis and to his original observations on diseases of the heart and lungs.

Neither Laennec nor his immediate followers paid any attention to changes in the pitch of percussion notes or respiratory sounds. Credit for this important addition to the art of physical diagnosis belongs to Austin Flint. His observations were embodied in an essay entitled, "The Variations of Pitch in Percussion and Respiratory Sounds, and Their Application to Physical Diagnosis." This essay was awarded the annual prize of the American Medical Association for 1852. Flint also won the first prize of the American Medical Association in 1859 for his essay, "The Clinical Study of the Heart Sounds in Health and Disease." This, too, was the year he published an important book on diseases of the heart.[1]

Flint's name is most frequently thought of in its association with the presystolic murmur which sometimes accompanies aortic regurgitation. This he was the first to describe. Flint heartily disapproved of associating any physical sign with the name of the original describer. He wrote:[2] "So long as signs are determined from fancied analogies, and named from these or after the person who describes them, there cannot but be obscurity and confusion."

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[1]Flint, Austin: *A Practical Treatise on the Diagnosis, Pathology, and Treatment of Diseases of the Heart*, Philadelphia, 1859, Blanchard and Lea, 473 pp.

[2]Landis, H. R. M.: Austin Flint: his Contributions to the Art of Physical Diagnosis and the Study of Tuberculosis. Bull. Johns Hopkins Hosp. 23: 182-186, 1912.

The first time he observed the so-called Flint murmur was in 1859, in examining a patient in Charity Hospital in New Orleans who had well-marked signs of aortic insufficiency and stenosis and in whom a presystolic murmur was audible at the apex At necropsy, however, the mitral valves were found to be normal This classic description was published in 1862 in the "American Journal of the Medical Sciences," and it is our privilege to reprint it here

Flint was by no means a specialist His contributions to the study of tuberculosis are among the best in American medical literature His first paper on this subject was published in 1849 In this early article Flint emphasized the importance of recognizing the disease in its incipient stages, for as he said, on this factor depended control of the disease

A masterful treatise on tuberculosis was his "Phthisis," published in 1873[3] The work is an analysis of 670 cases Landis said that it deserved to be ranked with the great work of Pierre C A Louis on tuberculosis

One of the distinguishing features of Austin Flint was the receptiveness of his mind to new ideas Shortly after Koch's momentous discovery of the etiology of tuberculosis in 1882, Flint began having the sputum of patients at Bellevue Hospital examined From his study of these patients he immediately saw the enormous importance of Koch's discovery In a paper[4] read in his seventy-second year, he emphasized the fact that tuberculosis could be contracted by a normal person's exposure to the disease

Flint's last article, which was published posthumously, was his "Medicine of the Future"[5] Therein he foresaw that physiologic and pathologic chemistry must be investigated to explain many of the phenomena of health and disease He had already accepted as valid the role of bacteria in health and disease and he foretold great progress in the understanding of this branch of medical science

Austin Flint's death was sudden and unexpected He had attended a meeting of the faculty of the Bellevue Hospital Medical College on Friday evening, March 12, 1886. On returning home he proceeded to retire and without any warning a cerebral hemorrhage occurred This was followed by unconsciousness, resulting in Flint's death fourteen hours later, on Saturday, March 13, 1886

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[3]Flint Austin *Phthisis, its Morbid Anatomy, Etiology, Symptomatic Events and Complications, Fatality and Prognosis, Treatment and Physical Diagnosis, in a Series of Clinical Studies*, Philadelphia, 1875 H C Lea, 446 pp

[4]Flint, Austin *On the Pathological and Practical Relations of the Doctrines of the Bacillus Tuberculosis*, 16 pp Reprinted from Med News Philadelphia, 1884, vol 440

[5]Flint Austin *Medicine of the Future* An address prepared for the annual meeting of the British Medical Association in 1886 New York 1886, D Appleton & Co, 37 pp

ON CARDIAC MURMURS

By

AUSTIN FLINT, M.D

Professor of the Principles and Practice of Medicine in the Bellevue Hospital Medical College, N Y, and in the Long Island College Hospital

THE clinical study of cardiac murmurs, within the last few years, has led to our present knowledge of the diagnosis of valvular lesions of the heart By means of the organic murmurs it is positively ascertained that lesions exist in cases in which, without taking cognizance of the murmurs, the existence of lesions could only be guessed at The absence of the organic murmurs, on the other hand, enables us generally to conclude with positiveness that valvular lesions do not exist As a rule, to which there are but few exceptions, these lesions may be excluded, if there be no murmur These are great results, but the practical auscultator of the present day need not be told that the clinical study of cardiac murmurs has led still further into the mysteries of diagnosis Having ascertained the different murmurs which occur in connection with valvular lesions, having traced their connection, respectively, with different lesions, having shown their relations to the movements of the several portions of the heart, and to the cardiac sounds,[1] and, having explained satisfactorily the mechanism of their production, we are able to determine not only the existence or non-existence of valvular lesions, but their particular situation when they are present, and, to a certain extent, their character and consequences The practiced auscultator, by listening to the murmurs alone, is able to tell whether lesions are situated at the mitral, or the aortic, or the pulmonic orifice, and he is able to say, in certain cases, that the valves which are to protect these orifices against a regurgitant current of blood, have been rendered by disease inadequate to their office It is unnecessary to adduce proof of these statements, their correctness is sufficiently known to those who are conversant with physical exploration as applied to the diagnosis of affections of the heart How strikingly do these facts exemplify the progress of practical medicine to those who, although still among the junior members of the profession, have practised before and since the recent developments in this department of our knowledge!

*Am J M Sc 44 29-54 1862

[1]The conventional distinction between the cardiac *sounds* and *murmurs* is to be borne in mind, the former term being limited to the normal heart-sounds with their abnormal modifications, and the latter to newly-developed or adventitious sounds, which are altogether the products of disease

These remarks are introductory to the consideration of various practical points pertaining to the cardiac murmurs And the first subject will relate to these murmurs in general—viz, *the limitations of their significance* After having considered certain points embraced in this subject, I propose to take up various points relating to the different murmurs separately

By the limitations of the significance of the murmurs, I mean the actual amount of knowledge respecting valvular lesions to be derived from this source It is evident, from what has been stated already, that the knowledge which they convey is of very great importance, but important as this knowledge is, it has certain limits which are not always sufficiently understood, and, as a consequence, the practitioner is liable to fall into unfortunate errors of opinion as regards the gravity of the lesions which the murmurs represent

Prior to the clinical study of the cardiac murmurs, the existence of organic affections of the heart was recognized when, in conjunction with disturbed action of the organ, symptomatic events had taken place which belong to an advanced stage of only a certain proportion of cases Dyspnoea, palpitation, and dropsy, were the symptoms mainly relied upon for the diagnosis The recognized cases were then comparatively rare, and, when recognized, a speedily fatal issue was expected This fact, together with the frequency with which cardiac lesions were revealed by postmortem examinations in cases of sudden death, rendered the diagnosis of organic disease of the heart equivalent to a summons from the grave The prognosis, as a matter of course, was as unfavourable as possible, the doom of the patient was either to die unexpectedly at any moment, or to endure protracted sufferings until released by death The study of the murmurs together with the application of other signs, enabled the practitioner to recognize organic affections at a period in the disease when otherwise they would not have been discovered The recognized cases became more frequent Persons were found to have cardiac lesions who presented few or no symptoms pointing obviously to disease of the heart The ideas which had prevailed relative to the gravity of organic affections, however, naturally enough, continued to prevail An organic murmur, consequently, had a fearful significance It was considered as proof of disease which was not less surely destructive because earlier ascertained Let it be said of a patient that he had a cardiac murmur denoting a valvular lesion, and his doom was pronounced, sudden death, which might occur at any time, or an early development of the distressing symptoms characteristic of cardiac disease, were to be expected, whatever might be his present condition

So far from concealing from patients the fearful significance of cardiac murmurs it was considered important for them to understand fully their precarious condition, in order to receive their co-operation in the meas-

ures of management which were deemed essential. These measures embraced general and local blood-letting, depletion by cathartics, sedative remedies addressed to the circulation, mercurialization, low diet, together with as much inaction of mind and body as possible. The consequences of this management were calamitous in the extreme. In fact, these measures contributed, in no small degree, to the fulfilment of the gloomy predictions impressed upon the minds of the unfortunate patients who were found to present the auscultatory sign of valvular lesions. So long as these notions with regard to the treatment of cardiac affections prevailed, an early diagnosis, instead of being desirable, was a serious disadvantage, and truly fortunate were they who kept aloof from the stethoscope of the auscultator!

Erroneous views respecting the significance of cardiac murmurs, and also respecting *the indications for treatment in cases of organic disease of* the heart, are still, to a greater or less extent, prevalent. I propose now to confine myself to the former, *i. e.*, the significance of the murmurs. It is obvious that *with the acquirement of means of ascertaining the* existence of lesions at an early period, when, without these means, the lesions could not have been discovered, clinical experience had to take a new point of departure as regards prognosis. And experience has shown that lesions giving rise to cardiac murmurs by no means invariably denote impending danger or serious evils, and that they are not unfrequently innocuous. Several clinical observers, within the last few years, have contributed facts going to show the correctness of this statement. Of these, Dr. Stokes[1] is especially prominent. Dr. Gairdner, of Edinburgh, has lately communicated a valuable paper on this subject.[2] I have been able to gather some facts having an important bearing on the subject under consideration. Of the cases which have come under my observation, exemplifying the "limitations of the significance of cardiac murmurs," I shall select a few of the most striking.

Thirteen years ago, I attended a child, aged 11 years, with a slight rheumatic attack. Directing attention to the heart, I found a very loud mitral, regurgitant murmur, heard over the left lateral surface of the chest and on the back. The heart was enlarged, the extent and degree of dulness in the praecordia being increased, and the apex beat without the nipple. The murmur was evidently not due to an endocarditis developed during the present attack of rheumatism, the lesion giving rise to it probably originated in an obscure thoracic affection which had occurred seven years before. I was at that time less acquainted with the significance of cardiac murmurs than now, and I deemed it my duty to inform the mother of the patient of the existence of an organic affection of the heart, which would be likely to destroy life within a period not

[1]Diseases of the Heart and Aorta.
[2]Edinburgh Monthly Journal of Med. Science.

very remote The patient is still living She is now 24 years of age, and, although presenting a delicate appearance, a casual observer would not suspect the existence of any disease She is subject to palpitation, to coldness of the extremities, and experiences want of breath on active exercise, but she does not consider herself an invalid, and the apprehensions caused by my communication to the mother have long since disappeared

It is fair to presume that my opinion in this case was considered as a mistake It was, indeed, an error of judgment as regards the prognosis, but the diagnosis was correct, the loud bellows murmur is still there, and heard over the whole chest, even through the dress, and the heart is considerably enlarged The patient, if not destroyed by some intercurrent affection, will ultimately die of cardiac disease, yet it is now twenty years since the probable commencement of the lesions giving rise to the cardiac murmur

Nearly twenty years ago a person was examined by a medical friend with reference to an assurance on his life My friend, finding a loud murmur, and an abnormally strong action of the heart, brought the person to me as an interesting case for examination I failed to record the case, and am not therefore positive as regards the particular murmur present, but I think it was the mitral regurgitant Since that examination, until recently, I have been in the habit of meeting this person often, although he has never been my patient He has been, and still is engaged in active business He is now about fifty years of age He has survived his wife, and been again married within a few years

I have selected these two cases as illustrating the duration of life and comfortable health for thirteen and twenty years after a loud organic murmur, together with enlargement of the heart, had been ascertained, in both cases life and comfortable health continuing at the present moment I could cite, in addition, numerous cases of persons now living, and apparently well, who have had organic murmurs for several years In making examinations of chests, supposed to be healthy, for purposes of study, I have repeatedly found a murmur, evidently organic, when no disease of the heart was suspected either before or after my examination The following case is instructive, as showing the importance of taking into account the coexistence of functional disorder of the heart, dependent on anaemia, with organic disease

In November, 1852, I visited, in consultation with Professor Rogers, of Louisville, a lady aged about 25 She had had repeated attacks of acute rheumatism She had an infant several months old, which she was nursing, and she had become quite anaemic She had begun to suffer from palpitation during the preceding summer, and her attention was attracted to a sound in the chest which she heard in the nighttime This sound was also heard by a sister with whom she slept She described, of her own accord, the sound to be like that produced by a pair of bellows

She had never heard of cardiac bellows-murmurs, and at this time there had been no examination of the chest. Prof. Rogers had been called to the patient a short time before my visit, and detected at once the existence of organic disease.

She presented an aortic direct and a mitral regurgitant murmur, both loud; the heart was moderately enlarged, and its action violent. She was conscious of this violent action, and slight exercise or mental excitement occasioned much distress from palpitation. The urgent symptoms in the case were attributed to anaemia; weaning was at once enjoined, and chalybeate remedies, etc., advised. I met the patient a year afterwards without recognizing her. She was apparently in perfect health, and presented a blooming appearance. Her friends thought we must have been mistaken in our opinion as to the existence of organic disease of the heart. The murmurs and the signs of enlargement, however, were still there. She continued to enjoy good health until the summer of 1856, when she suffered from uterine hemorrhage, and again became anaemic. The action of the heart became irregular, and she complained much of vertigo. Tonics, stimulants, nutritious diet and fresh air failed to remove the anaemic state, and at length she was seized with apoplexy and hemiplegia. She recovered from the apoplexy, but the hemiplegia continued, and death took place between two and three weeks after the apoplectic seizure.

The significance of organic murmurs is limited to the points of information already stated in the introductory remarks, viz., the existence of lesions, their localization, and the fact of valvular insufficiency or regurgitation. Whether the lesions involve immediate danger to life, or, on the contrary, are compatible with many years of comfortable health, the murmurs do not inform us, nor do they teach us how far existing symptoms are referable to the lesions, and how far to functional disorder induced by other morbid conditions. Neither the intensity nor the quality of sound in the murmurs furnish any criteria by which the gravity of the lesions or their innocuousness can be determined. A loud murmur is even more likely to be produced in connection with comparatively unimportant lesions than with those of a grave character, because in the former, rather than in the latter case, is the action of the heart likely to be strong, and the intensity of the murmur, other things being equal, will depend on the force with which the currents of blood are moved. Whether the murmur be soft, or rough, or musical, depends not on the amount of damage which the lesions have occasioned, but on physical circumstances alike consistent with trivial and grave affections.

It may be imagined that these assertions, although true as regards murmurs produced by the direct currents of blood, do not hold good with respect to the regurgitant murmurs. The latter, it may be said, involving as they do insufficiency of the valves, will be loud in proportion

to the amount of blood which regurgitates, and, therefore, the intensity of the murmur should be a criterion of the amount of valvular insufficiency. But clinical observation disproves this surmise. A minute regurgitant stream is as likely to be intensely murmuring as a large current, perhaps even more so. Here, too, the loudness of the sound will depend, in a great measure, on the power of the heart's action. To this point I shall recur when I come to consider the different murmurs separately.

The practical injunction to be enforced in connection with the limitations of the significance of the cardiac murmurs is, that we are not to judge of the magnitude of valvular lesions, of the amount of danger on the one hand, or of the absence of danger on the other hand, by the characters belonging to the murmurs. The physician who undertakes to interrogate the heart by auscultation is not to decide that the condition of his patient is alarming, simply because he finds a murmur which he satisfies himself is dependent on an organic lesion of some kind. The lesion may be at that time, and perhaps ever afterwards, innocuous; the evils arising from cardiac affections may be remote, and so far from plunging the patient into despair by the announcement of the fact that he has an incurable disease of the heart, there may be just grounds for holding out expectations of life and comfortable health for an indefinite period. Neither does it necessarily alter the case when more than one murmur is discovered; the existence of several murmurs by no means excludes the possibility of similar encouragement. We are to look to other sources of information than the murmurs in forming an opinion respecting the gravity of the affection. What are the sources of information on which our opinion is based? It does not fall within the scope of this essay to consider at length the points involved in the answer to this inquiry. I shall answer it in a few words.

The heart-sounds furnish means of determining whether the lesions are of a nature to affect materially the function of the valves. I must here pass by this useful and beautiful application of auscultation with a simple allusion to it, referring the reader elsewhere for a full exposition of the subject.[1] I shall, however, return to the subject presently in considering the murmurs individually. Means requiring less proficiency in physical exploration relate to enlargement of the heart. It is not a difficult problem to determine whether the heart be or be not enlarged, and it is easy to determine the degree of enlargement. Now, in general, if valvular lesions have not led to enlargement of the heart, they are not immediately dangerous, and the danger is more or less remote. Here is a criterion of great importance in estimating the gravity, on the one hand, or the present innocuousness on the other hand, of lesions giving rise to murmurs. So long as the heart be not enlarged, the lesions cannot have occasioned to much extent those disturbances which arise from contrac-

[1]Clinical Study of the Heart-sounds in Health and Disease, Prize Essay, Trans. Amer. Med. Association, 1859.

tion or patency of the orifices The murmurs, in themselves, give no information respecting the amount of obstruction from contracted orifices, or of regurgitation from valvular insufficiency Let this fact be constantly borne in mind But obstruction and regurgitation, singly or combined, inevitably lead to enlargement of the heart, hence the latter becomes evidence of the former The degree of enlargement is, in general, a guide to the extent and duration of the disturbances occasioned by contracted and patescent orifices As a rule, if the heart be slightly or moderately enlarged, the immediate danger from the lesions which may give rise to one or more loud murmurs is not great

The truth is, the evils and danger arising from valvular lesions, for the most part, are not dependent directly on these lesions, but on the enlargement of the heart resulting from the lesions We may go a step further than this and say that, ordinarily, serious consequences of valvular lesions do not follow until the heart becomes weakened either by dilatation or by degenerative changes of tissue So long as the enlargement be due mainly to hypertrophy of the muscular walls, the patient is comparatively safe Hypertrophy is a compensatory provision, the augmented power of the heart's action enabling the organ to carry on the circulation in spite of the disturbance due to obstruction and regurgitation Happily, in most cases, hypertrophy is the first effect of valvular lesions, and, for a time, it keeps pace with the progress of the latter Dilatation which weakens the heart's action, is an effect consecutive to hypertrophy, and, as a rule, it is not until the dilatation predominates that distressing and dangerous evils are manifested

The practical bearing of these views respecting hypertrophy and dilatation, on the management of organic affections of the heart, is obvious They are inconsistent with the employment of measures to prevent or diminish hypertrophy, on the contrary, they point to the importance of an opposite end of management, viz, to encourage hypertrophy in preference to dilatation, and to maintain the vigour of the heart's action It does not fall within the scope of this essay to consider therapeutical applications, and I must content myself with this passing notice of an immensely important reform in the management of organic affections of the heart

Returning to the means of determining the gravity of valvular lesions, I repeat, they become serious, in other words, the distressing and dangerous symptomatic events are to be expected, in proportion as hypertrophy merges into dilatation, or as weakness of the organ may be induced by structural degeneration or other causes In connection, then, with murmurs, we are to determine the condition of the heart as respects the points just mentioned, in order to estimate properly the gravity of the lesions which the murmurs represent In leaving this subject, viz,

the limited significance of the cardiac murmurs, I will give a case which is a type of a class of cases not infrequently coming under observation

In the spring of 1860, I was consulted by a medical gentleman from a distant State, who furnished me with the following written statement of his case —

"About a year ago I went to the city of ——— to place myself under the care of Dr ———, for a trifling surgical difficulty with which I had been annoyed for a long time At long intervals previous to that time I had had severe pains in the left breast about the cardiac region, but at no time from any constant pain I thought the pain was of a neuralgic character While at ——— I thought I would have my lungs examined, as some members of my family had been consumptive I went to Dr ——— and to Dr ———, both of whom pronounced my lungs sound, but said that my heart was affected I came home much depressed by their opinion, and suffered so much from mental anxiety that in the course of a month or two I determined to go back and consult another medical gentleman, Dr ——— He told me there was some roughness about the sounds of the heart but no serious organic disease I was much relieved by this opinion, and clung to the belief that the pains were of a neuralgic character[1] Previous to my going to ——— I had all my life taken a good deal of out-door exercise, such as riding, hunting, fishing, etc, for the purpose of warding off any tendency to consumption I have always had a frail figure and been inclined to despondency I have suffered a great deal of anxiety, owing to family affairs and business matters After my return from consulting Dr ——— I thought it best to give up active exercise for fear of increasing any cardiac affection that might exist I do not think that I have had any severe pain in my chest frequently, at any time, but only at intervals and apparently occasioned by anxiety about patients, etc

"In December last I went into the country, 13 miles to see a patient The weather was very cold, rainy, and windy, I returned in the night I was suffering from toothache and smoked a cigar in order to relieve the pain I went over to my office to write a prescription for a sick child, and on my way back I was attacked by palpitation of the heart for the first time in my life I came into the house and lay down, when I was seized with severe rigors without chills I had also pain in the back, and afterwards fever Since then I have been subject, at intervals, to a jarring or knocking sensation about the heart, but no palpitation of long continuance I cannot sleep as well on my left side as formerly, as it causes an uneasy sensation with something like palpitation and some pain I do not take much exercise, and find that I get out of breath easily I am very sensitive to cold The attacks of increased action of the heart are always accompanied by rigors and irritability of the bladder On the 19th of March, I was taken with a feeling of fatigue and indigestion, followed by severe rigors together with great heat of the head and body The circulation was rapid and accompanied by palpitation The attack lasted nearly an hour, and I feel the effect of it today, March 22d I notice, when reading a newspaper or small book, that the action of the heart causes it to vibrate During my first attack in Dec, I had

[1]Doubtless they were so

an intermittent pulse. I did not recover from that attack so as to go out for a week, and have not since been as well as before.

"Fearing that my situation was critical I have been careful of myself. I have feared to increase the affection and that I might die suddenly. But I have had fear that in taking care of the cardiac affection I shall increase a tendency to consumption. Any mental anxiety increases the action of the heart. I do not smoke nor chew tobacco, nor drink any alcoholic liquors. I have suffered much from toothache; in other respects have had generally very good health. I have never had rheumatism. I am a married man with five children. I think my cardiac affection has been getting worse since December last, and I suffer in mind dreadfully on that account, as I have a great deal to live for."

On examination of the chest, in this case, I found the apex-beat in the 5th intercostal space half an inch within a vertical line passing through the nipple. The area of superficial cardiac dulness carefully delineated on the chest, was found to be of normal extent. The left border of the heart fell within the nipple. The respiratory murmur, on a deep inspiration, was heard over the whole praecordia. The apex-beat was not abnormally strong; no other impulse was discovered, and no heaving of the praecordia.

At the first examination, the heart being but little excited, I discovered a slight murmur just to the left of the apex, heard only during the latter part of each inspiratory act. I could discover no murmur at the base. At a subsequent examination on the same day, made after dinner, the patient having drank a little wine with his dinner, the action of the heart was much greater than at the previous examination. I then discovered a well-marked systolic murmur at the apex, to the left of the apex and at the lower angle of the scapula; I also ascertained the existence of a soft systolic murmur at the base on the left side of the sternum and not on the right side. This murmur extended over the whole summit of the chest on the left side. At the summit it came evidently from the subclavian, as the pitch differed from that of the murmur over the pulmonary artery, *i.e.*, in the 2d intercostal space on the left side.

On the next morning I made an examination while the patient was still in bed. The heart was then acting tranquilly. I discovered a feeble murmur at the apex only; this murmur was not perceived behind, and no murmur was heard at the base.

The aortic and pulmonic second sounds were normal, and so also were the mitral and tricuspid valvular elements of the first sound.

I shall quote from my record book the remarks which were appended to this case when the record was made:—

"The heart is but little if at all enlarged, and the heart sounds are normal. There exist, therefore, no lesions which at present are of serious import. The cardiac trouble which has occasioned the patient so much unhappiness and anxiety, is purely functional.

"Dr ——— (who first examined this patient) evidently discovered a murmur His examination was not very critical, and was made after the patient had just mounted stairs at his hotel The opinion that there was organic disease without any qualifying explanations produced a profound moral impression on the patient The opinion of Dr ——— subsequently did something toward revealing the apprehensions of the patient, but his coming such a long distance to consult me is evidence how much his mind was ill at ease on the subject

"The heart is not entirely free from lesions, there is slight mitral regurgitation The murmur at the base is perhaps inorganic, or at all events it does not denote important valvular lesions, since a comparison of the aortic and pulmonic sounds show the two to be in a normal relation to each other The lesions in fact which exist in the case are of no immediate seriousness, and of this I assured the patient in the most positive manner

"This case affords an illustration of the importance of discriminating between functional disorder and the effects of organic disease when there is evidence of the latter It illustrates, also, the importance of the heart sounds and of the size of the heart in determining the gravity of lesions The evils which may arise from the lesions (if they ever occur) are remote, and I felt warranted in assuring the patient that his condition involved no present danger, and that he might dismiss all thoughts of disease of the heart I ordered him to live well and to resume his out-door sports His apprehensions were entirely relieved by my assurances, and his expressions of gratification afforded evidence of what he had suffered mentally from the idea of an organic disease incapacitating him from the duties of life and rendering him liable to sudden death "

As I have said, this case is a type of a class of cases of not infrequent occurrence The existence of a cardiac murmur was discovered in consequence of an examination with reference to the lungs Prior to this time no symptoms of disorder of the heart existed, the discovery of the murmur was an unfortunate circumstance for the patient, the belief that he had serious disease of the heart became fixed in his mind, and doubtless contributed to the disorder which subsequently occurred The functional disorder was slight in comparison with cases which are of daily occurrence, but the patient naturally attributed it to organic disease The affection was in fact altogether functional, albeit the existence of an organic murmur, this is the practical point which the case is intended to illustrate

I propose now to consider certain practical points pertaining to the cardiac murmur separately, I shall limit my remarks mainly to the murmurs produced by the blood-currents, in the left side of the heart, viz, the *aortic direct, the aortic regurgitant,* the *mitral systolic* and the *mitral direct* Exclusive of the *pulmonic direct* murmur I have but little practical acquaintance with murmurs emanating from the right side of the heart

Aortic Direct Murmur —The question whether a murmur be organic or inorganic has reference generally to a murmur produced by the current of blood from the left ventricle into the aorta The aortic regurgitant

murmur and a mitral murmur which is truly regurgitant are of necessity organic, they require lesions involving more or less insufficiency of the valves The mitral direct murmur, as will be seen presently, is inorganic only as a rare exception to the rule A practical point, then, in certain cases, is to determine whether an existing aortic direct murmur be organic, *i e*, dependent on lesions, or whether it be inorganic, *i e*, dependent on a blood change This point cannot always be positively settled, but when such is the case it is practically not very important that it should be settled, in other words, when a murmur exists concerning which we are at a loss to decide whether it be organic or inorganic, if it be the former, the lesion giving rise to it must be trivial, since under these circumstances the heart sounds will be found to be normal and the heart not enlarged If in connection with an aortic direct murmur we find the aortic second sound impaired and the heart enlarged, we are warranted in considering the murmur organic But a slight rippling of the current by roughening from an atheromatous or calcareous deposit which occasions no obstruction, and no valvular insufficiency, may yield a murmur How are we to distinguish this from an inorganic murmur? The absence of the anaemic state, of other cardiac murmurs, of arterial murmurs, of the venous hum, and the persistency and uniformity of the murmur are the circumstances which render it probable that it is organic, while the existence of anaemia, of other cardiac murmurs, of arterial murmurs and the venous hum, together with intermittency and variableness of the murmur, render it probable that it is inorganic

In my work on diseases of the heart, 1859, I have stated roughness of the murmur to be one of the circumstances showing it to be organic I then believed that an inorganic murmur was never rough The able reviewer of my work in the *Dublin Quarterly* says, with regard to this point, "We are unable to give unqualified assent to the statement that an inorganic murmur is uniformly soft" The criticism of the reviewer is just, I was mistaken in the statement as the following case will show —

I visited in May, 1860, a female patient who presented a loud rasping murmur which had led to the suspicion of aneurism The patient was exceedingly anaemic, there was total loss of appetite with vomiting and diarrhoea The anaemia could not be accounted for, it belonged in the category of cases described by Addison as cases of idiopathic anaemia I found a rough rasping murmur at the base of the heart on the right of the sternum, and a similar murmur was heard over the subclavian and carotid On examination after death, in this case, the heart was perfectly normal, the aortic orifice, the aorta, subclavians, and carotids were free from any morbid change, and the lungs were healthy The murmur was evidently due to a blood change

The discrimination of an aortic direct from a pulmonic direct murmur is a point of interest If the normal situation of the aortic and pulmonic

artery in relation to the walls of the chest be preserved, an aortic direct murmur has its maximum of intensity and may be limited to the point where the aorta is nearest the surface, viz, the second intercostal space on the right side close to the sternum But the normal relation of the vessels to the thoracic walls is not infrequently changed when the heart becomes enlarged, or as a consequence of past or present pulmonary disease, and hence this murmur may be loudest or limited to the base on the left side of the sternum The situation of the murmur or of its maximum, therefore, is not always reliable in the discrimination A pulmonic direct murmur has its maximum or is limited to the second or third intercostal spaces on the *left* side close to the sternum, the artery being at these points nearest the surface, but, as just stated, an aortic direct murmur may be found to be loudest in this situation If the heart be not enlarged or displaced by pressure from below the diaphragm, the chest not depressed, and the lungs are free from disease, the fact that a murmur has its maximum at or is limited to the right side of the sternum, is evidence of its being aortic rather than pulmonic, and *per contra,* the fact of a murmur having its maximum at or being limited to the left side of the sternum, is evidence of its being pulmonic rather than aortic But the propagation of the murmur into the carotid is the most important circumstance in this discrimination An aortic direct murmur, unless it be quite weak, is generally propagated into the carotid A pulmonic direct murmur of course cannot be Here attention to the pitch and quality of sound is called into requisition It is to be determined that a murmur heard over the carotid is propagated from the aorta not produced within the carotid How is this to be determined? Very easily in most cases, by a simple comparison of the murmur as heard over the carotid and at the aortic orifice If the murmur in the neck be a propagated murmur it will differ from that at the base of the heart chiefly as regards intensity, the pitch and quality will not be materially changed If it be rough or soft at the base of the heart, it will be the same in the neck, if the pitch be high or low at the base of the heart, it will be the same in the neck On the other hand, a murmur produced within the carotid, will be likely, in the great majority of cases to differ in quality and pitch from a coexisting murmur at the aortic orifice

In accordance with what has been stated with reference to the limitations of the significance of organic murmurs in general, an aortic direct murmur, when undoubtedly organic, alone affords little or no information respecting the nature and extent of the lesions which give rise to it A comparison of the aortic with the pulmonic second sound of the heart enables us frequently to form an opinion as regards the amount of damage which the aortic valve may have sustained The aortic second sound, in health, as heard in the right second intercostal space near the sternum, is more intense, and has a more marked valvular quality, than

the pulmonic second sound as heard in a corresponding situation on the left side. Now, it is often easy to determine whether the intensity of the aortic second sound is diminished and its valvular quality impaired, and in proportion as this sound is abnormally altered in these respects, we may infer that the aortic valve is damaged. It is hardly necessary to say that, in order for this comparison to warrant the inference just stated, pulmonary disease must be excluded. A tuberculous deposit, for example, on the left side, may, by conduction, render the pulmonic apparently more intense than the aortic sound, the latter retaining its normal intensity; the same will occur from shrinking of the upper lobe of the left lung so as to bring the pulmonary artery into contact with the thoracic walls. Under the latter circumstances the pulsation of the pulmonic artery may sometimes be distinctly felt in the second left intercostal space near the sternum. I have met with two cases during the past winter in which the pulsation of the pulmonic artery was so strong as to suggest the idea of aneurism; in both cases the patients were affected with tuberculous disease of the left lung. Alteration of the normal relation of the aorta and pulmonic artery due to enlargement of the heart, or to any of the causes already mentioned, will of course preclude a comparison of the two sounds.

With reference to the value of a comparison of the aortic and pulmonic second sound in estimating the amount of aortic lesions, the able reviewer in the *Dublin Quarterly*, to whom I have already referred, and for whose valuable criticisms I beg to avail myself of this opportunity of expressing my sincere thanks, remarks as follows:—

> "It is observed, to our great wonder, that if the aortic second sound retain its normal intensity and purity, it shows that the aortic valve is competent to fulfil its function, *a fact which warrants the exclusion of lesions affecting it sufficiently to give rise to obstruction.*" He adds, "Surely Dr. Flint must have become clinically cognizant of the fact that there is not unfrequently serious contraction of the aortic orifice producing marked obstruction and hypertrophy of the left ventricle, the aortic second sound remaining intact."

This criticism is not altogether just. I state that the normal intensity and purity of the aortic second sound warrant the exclusion of lesions affecting it, *i. e.*, the valve, sufficiently to give rise to obstruction. I do not say that contraction of the aortic orifice may not occur without involving the aortic valve, and, in such a case, the aortic second sound may remain intact. In fact, I imply this when I proceed to say, "In a large proportion of the cases of obstructive lesions of the aortic orifice, the valve is involved sufficiently to compromise, to a greater or less extent, its function and impair the intensity of the aortic second sound." This language is equivalent to admitting that there is a small proportion of cases of obstructive lesions of the aortic orifice, in which the valve is *not* involved

sufficiently to compromise its function and impair the intensity of the aortic second sound These exceptional cases are extremely rare Surely the able reviewer will admit that, in the great majority of cases, the valve is involved so as to impair its function to a greater or less extent

I have lately been interested in a nice point of observation connected with the murmur under consideration, viz, the concurrence of two aortic direct murmurs, one produced at the aortic orifice and another within the aorta just above the orifice One of the murmurs may be organic and the other inorganic, or both murmurs may be organic At the present moment I have under observation three cases of endocarditis with rheumatism, each presenting a high pitched basic murmur when the stethoscope is placed over the sternum and a little to the right of the median line, the murmur limited to a circumscribed space, and just above this point, in the right second intercostal space, is another murmur differing from the former notably in pitch, being quite low In one of these cases there is still another murmur in the pulmonic artery The high pitched murmur just below the second intercostal space, as I infer from the situation to which it is limited, is a murmur produced at the aortic orifice, and the low pitched murmur just above, as I infer, also, from the situation to which it is limited, is an aortic murmur produced within the artery above the aortic orifice I infer that there are two murmurs from the notable difference in pitch, it being by no means probable that a single sound would be so much altered within the area in which the two murmurs are heard, this area not being larger than a half dollar That an aortic murmur is sometimes produced at the orifice and sometimes within the artery above the orifice, in different cases, is certain, but I am not aware that the production of a murmur in each situation, at the same time, in the same case, and the discrimination of the two by means of the character of the sound, have been pointed out

Aortic Regurgitant Murmur —This murmur need never, as a matter of course, be confounded with the systolic murmurs, viz, the aortic direct, and mitral regurgitant, the latter occurring with the first, and the former with the second sound of the heart In general, too, there is no difficulty in distinguishing the aortic regurgitant, from the mitral direct murmur The former occurs with and follows the second sound, the latter precedes the first sound The one is diastolic, the other is pre-systolic This is a distinction, nice, it is true, but easily appreciable in practice, to which I shall recur under the heading of the mitral direct murmur

The situation of the murmur is also distinctive It is best heard at, and below the base of the heart Usually it is best heard below the base to the left of the median line on a level with the third or fourth ribs This is doubted by the reviewer in the *Dublin Quarterly*, to whom I have referred, but as the statement is based on a pretty large number of recorded observations, I must consider it as correct It is not uncommon to hear

this murmur distinctly, and even loudly, over the apex, it may be diffused over the whole præcordia and even propagated beyond this region.

An aortic murmur with the second sound of the heart, propagated below the base of the heart, necessarily implies regurgitation, in other words there must be insufficiency of the aortic valvular segments. But it is always to be borne in mind that no inference can be drawn from the intensity or character of the murmur, respecting the amount of insufficiency and consequent regurgitation. An extremely small regurgitant stream may give rise to a loud murmur, while a feeble murmur may accompany a large regurgitant current, as the rippling brook is noisy while the deep broad river flows silently. In a case recently under observation, there existed a loud aortic regurgitant murmur, and on examination after death the aortic segments were so slightly impaired that, on cursory inspection, they might have been considered as normal. Weakening or extinction of the aortic second sound of the heart are points of importance as showing frequently the extent to which the function of the aortic valve is impaired. Comparison with the pulmonic sound enables us to judge whether the aortic sound be impaired, provided the pulmonic sound be not abnormally intensified as a result of coexisting mitral lesions. It is important to recollect that when aortic and mitral lesions coexist, the intensity of the pulmonic sound cannot be taken as a criterion for judging whether the aortic sound be, or be not weakened. This remark is equally applicable to the comparison in cases in which an aortic direct murmur is present. It is needless to say that in comparing the aortic and pulmonic sound in connection with an aortic regurgitant, as with an aortic direct murmur, pulmonary disease is to be excluded, *i e*, solidification or shrinking of the left lung will, as already stated, render the pulmonic sound relatively more intense than the aortic, irrespective of, on the one hand, any actual increase of the intensity of that sound, or, on the other hand, of any weakening of the aortic sound. It is also to be stated here, as heretofore, that an alteration of the situation of the aorta and pulmonary artery as regards the thoracic walls, due to enlargement of the heart, or other causes, will preclude a comparison of the two sounds with reference either to intensification of the pulmonic, or weakening of the aortic sound.

Mitral Systolic Murmur.—I use the phrase mitral *systolic,* instead of that more commonly used, viz, mitral *regurgitant* murmur, as applied to any murmur produced at the mitral orifice and accompanying the first sound of the heart. If the latter term be applied to any systolic murmur emanating from the mitral orifice, we fall into the solecism of calling a murmur regurgitant in cases in which there is no regurgitation. A mitral murmur may be produced by mere roughness of the valvular curtains when there is no insufficiency of the valve. In this case the murmur cannot be correctly said to be regurgitant. A mitral systolic murmur, thus, may or may not be a regurgitant murmur, and, to express this important

distinction, we may say that a mitral systolic murmur exists with or without regurgitation The question at once arises, how are we to determine whether a mitral systolic murmur be regurgitant or non-regurgitant? This point claims consideration

A mitral systolic murmur, as is well known, generally has its maximum of intensity at, and the murmuring may be limited to, the situation of the apex-beat, or to the point where the intensity of the first sound of the heart is greatest The murmur may be diffused, in the first place, within this point over the body of the heart, and, in the second place, without the apex over the left lateral surface of the chest and on the back I have been led to believe that when the murmur is diffused over the left lateral surface and more or less over the back, it always denotes regurgitation, and that when the murmur is not propagated much without the apex, although it may be more or less diffused over the body of the heart, it may be produced within the ventricle and not by a regurgitant current In the latter case I have distinguished the murmur as an intra-ventricular murmur, and not considered it as affording any evidence of insufficiency of the mitral valve It is this intra-ventricular, or mitral systolic non-regurgitant murmur, which generally exists in rheumatic endocarditis The importance of the point involved is obvious, for a murmur emanating from the mitral orifice without valvular insufficiency or regurgitation, denotes lesions of little immediate consequence, and they may be innocuous, not only for the present but for the future

The practical rule just stated, I believe, generally holds good, but there may be exceptions The following is perhaps an exceptional instance A case was recently under my observation in Bellevue Hospital, in which acute rheumatism was complicated with endocarditis, pericarditis, and pleurisy, with considerable effusion, affecting the left side The patient presented, on admission, a loud pericardial friction sound diffused over the whole praecordia, and a loud mitral systolic murmur The latter had its maximum of intensity at the apex, but was diffused over the left lateral surface of the chest and heard on the back After the lapse of about a week the friction sound disappeared, but before the disappearance of the friction sound, the endocardial murmur had gradually diminished and disappeared The pleuritic effusion also disappeared, and evidence was afforded in this case of pericardial adhesions by the immobility of the apex-beat when the body of the patient was placed in different positions The disappearance of an endocardial murmur developed by rheumatic endocarditis, so far as my observation goes, is rare, although I have met with other examples I suppose that endocarditis does not involve actual regurgitation save as a remote consequence of lesions to which the endocarditis may give rise I may be mistaken in this supposition, but, assuming that I am not, here was an instance in which

an intra-ventricular or non-regurgitant mitral systolic murmur was propagated entirely around the chest

With reference to determining the existence of either regurgitation or obstruction, or both, resulting from mitral lesions, a comparison of the aortic and pulmonic second sound, forms a beautiful and useful application of auscultation Obstructive and regurgitant lesions, situated at the mitral orifice, involving an obstacle to the free passage of blood through the pulmonary circuit, give rise, as is well known, to hypertrophy of the right ventricle In this way they lead to intensification of the pulmonic second sound of the heart This effect is due, in part, to the augmented power of the contractions of the right ventricle, and, in part, to the resistance to the passage of blood through the lungs, both continuing to increase the dilatation of the pulmonary artery by the pulmonic direct current, and the consequent recoil of the arterial coats by which the pulmonic valvular segments are expanded, and the pulmonic second sound produced But the morbid disparity between the aortic and pulmonic second sound is due, not alone to the intensification of the latter in the manner just stated The aortic second sound is weakened in proportion to the amount of blood which fails to pass into the aorta with the ventricular systole, in consequence of the mitral obstruction or regurgitation It is obvious that the aortic direct current will be lessened by the amount of blood which, in consequence of valvular insufficiency, flows backward into the left auricle after the ventricle contracts, and by the amount of difficulty which exists in the free passage of blood from the auricle into the ventricle in consequence of a contracted orifice It is also obvious that, other things being equal, the intensity of the aortic second sound will be greater or less according to the quantity of blood propelled into the aorta by the ventricular systole Thus, it is clear how mitral obstruction and regurgitation lead to weakening of the aortic sound, as well as to intensification of the pulmonic sound, and both effects are abundantly attested by clinical observation

The degree of weakening of the aortic and of intensification of the pulmonic sound will be proportionate to the amount of mitral regurgitation or obstruction, or both We have then, in this application of auscultation, a means of obtaining information respecting the extent or gravity of mitral lesions And, in a negative point of view, this application is important, viz , as a means of determining that lesions which give rise to a murmur are not serious, in other words, of determining that they do not involve much, if any, obstruction or regurgitation As enabling us to exclude obstructive or regurgitant lesions in certain of the cases in which mitral murmurs exist, a comparison of the aortic and pulmonic sound is of great practical value But the circumstances which may stand in the way of this application of auscultation are to be borne in

mind The two sounds cannot be compared with reference to mitral, more than with reference to aortic lesions, if there be coexisting pulmonary disease, nor whenever the normal relation of the aorta and pulmonary artery to the thoracic walls is altered by either past or present disease of the lungs, by deformity of the chest, or any other cause It is also to be recollected that mere enlargement of the heart may disturb the normal relation of these vessels to the walls of the chest This application, moreover, cannot be made when mitral and aortic lesions coexist Under the latter circumstances it is, of course, difficult or impossible to determine how far an existing disparity between the aortic and pulmonic sound is due to the aortic, and how far to the mitral lesions

Another important point pertaining to a mitral systolic murmur is, its occurrence without any appreciable lesions A truly mitral regurgitant murmur doubtless always involves lesions of some kind, for it is hardly probable that the papillary muscles, as has been supposed, may become spasmodically affected and thus give rise to insufficiency or regurgitation as a temporary functional disorder But it is undoubtedly true that a systolic murmur either limited to, or having its maximum of intensity near the apex, has been repeatedly observed in cases in which mitral lesions were not apparent after death Dr Bristowe in a paper contained in the *Brit and For Med Chir Review*, for July, 1861, details six cases of this description Dr Barlow, in an article in *Guy's Hospital Reports*, vol v, 1859, states that a mitral murmur may occur (for what reason he does not state) in long-continued capillary bronchitis I have met with some instances in which a systolic murmur, supposed to be mitral, existed, and no mitral lesions were found after death

Case 1[1] In the winter of 1859-60, I saw a female patient in the Charity Hospital, New Orleans, in the service of my colleague, Prof Brickell, affected with capillary bronchitis After several days there was improvement as regards the pulmonary symptoms, and then, for the first time, a systolic cardiac murmur was discovered The murmur was loudest at the epigastrium, but heard over the site of the apex, and extended to, but not above the base of the heart The patient subsequently died On examination after death the lungs were emphysematous, there were no valvular lesions, all the valves appearing to be sound The foramen ovale was closed There were no clots The right ventricle was distended with liquid blood The walls of the heart were of normal thickness The valves and orifices were not measured, nor was the water test of valvular sufficiency employed

In recording this case I have commented on the murmur as follows ' What could have caused the loud systolic murmur? I cannot say unless it was due to distension of the right ventricle and tricuspid regurgitation "

[1]Private Records vol xi p 36

In support of the supposition that the murmur was tricuspid, not mitral, it is to be noted that the greatest intensity was at the epigastrium It was, however, considered to be a mitral systolic murmur during life

Case 2[1] During the winter of 1860-61, a patient was under my observation in the Charity Hospital, New Orleans, for four months, affected with albuminuria and general dropsy During all this time there was a mitral systolic murmur at the apex and over the body of the heart, and not propagated without the apex It was regarded as a mitral systolic, nonregurgitant or intra-ventricular murmur, and as such pointed out to several private classes in auscultation The patient died by asthenia, and was found to have fatty kidneys and cirrhosis of the liver On examination of the heart, *post mortem,* nothing abnormal was found except some enlargement, the organ weighing 12 oz, and a little separation of the marginal extremity of two of the aortic segments The mitral valve appeared to be perfectly normal I expected to find some roughening of the mitral valve but no insufficiency, there was, however, no atheromatous, calcareous or other deposit, and the valve seemed to be sufficient There was no aortic, nor pulmonic murmur in this case, a fact which excludes the supposition that the existing murmur was due to the condition of the blood

Case 3[2] During the winter of 1860-61, a patient was under my observation in the Charity Hospital, New Orleans, for about six weeks, affected with chronic bronchitis and emphysema of lungs He presented habitual dyspnoea which was at times excessive, persisting lividity and anasarca The heart was evidently somewhat enlarged There was a loud rough systolic murmur, having its maximum of intensity at the apex propagated without the apex (the record does not state how far), and over the body of the heart On examination after death the volume of the heart was not much increased, and its weight was 13 oz The left ventricle was not dilated and the left auricle was small The walls of the left ventricle did not exceed half an inch in thickness, and the appearance of the muscular tissue was healthy The mitral valve was perfectly normal The orifice was not enlarged, and the valve must have been sufficient No lesion at the aortic orifice The right cavities were much dilated They were twice as large as the left cavities The walls of the right ventricle were much thickened, the thickness falling but little short of that of the left ventricle No lesion of the pulmonic orifice The tricuspid valve was normal The orifice was very large, admitting the extremities of all the fingers I have appended to the record of this case the following comment "Whence the murmur supposed to be a mitral regurgitant? I suspect it was a tricuspid regurgitant "

Dr Bristowe, in the article already referred to, discusses several conditions which have been supposed to give rise to the murmur in cases like those which have just been given, viz, clots in the ventricular cavity,

[1]Private Records vol xi p 243
[2]Hospital Records vol xv p 423

spasm of the papillary muscles, and enlargement of the auricular orifice so as to render the valve insufficient. His own opinion is that the murmur is due to a "disproportion between the size of the ventricular cavity and the length of the chordae tendineae and musculi papillares." This disproportion he attributes to dilatation of the cavity of the ventricle. He also accepts to some extent an explanation offered by Dr. Hare, viz., that the murmur may be due to a "lateral displacement of the origins of the musculi papillares in consequence of the rounded form which dilatation imparts to the heart."

These several explanations may each be applicable to certain cases, but none of them, apparently to the cases which I have given. Clots in the left ventricular cavity were not present in either of the cases, the murmur continued too long and too persistently to be due to spasm, the mitral orifice was not dilated, and the enlargement of the heart was not sufficient to occasion a notable disproportion between the length of the tendinous cords and papillary muscles, and the ventricular cavity. I am disposed to think that in each of the three cases the murmur was erroneously considered to be mitral, that it was a tricuspid regurgitant murmur. As I have already said, I have but little practical knowledge of tricuspid murmurs. I have met with two instances in which murmur was connected with well-marked tricuspid lesions as verified by examination after death. In both these cases the murmur was heard over the body of the heart, within the superficial cardiac region. I suspect that a tricuspid regurgitant murmur is not so rare as is generally supposed, and that not very infrequently it is considered to be mitral. This opinion is expressed by Dr. Gairdner in an interesting article on cardiac murmurs in the *Edinburgh Med. Monthly*, Nov., 1861. According to this able clinical observer, a tricuspid systolic murmur is heard over the right ventricle where it is uncovered of lung, being but slightly audible above the third rib, and, if the heart be much enlarged, it may be heard louder towards the xiphoid cartilage. A collection of clinical facts respecting the frequency of tricuspid murmurs, the physical conditions giving rise to them, and the means of discriminating them from mitral murmurs, is an important desideratum.

Mitral Direct Murmur.—This murmur is not recognized by many auscultators, and its existence is denied by some. It is generally confounded with a mitral systolic murmur. For many years after I had begun to devote special attention to cardiac affections, I committed this mistake, and I was sometimes puzzled to account for a supposed mitral systolic murmur rough at its beginning and soft at its ending. In my records of some cases before I had learned to separate the mitral direct from a mitral regurgitant, I have described the latter as presenting the variation just stated, the fact being that the two murmurs were present, the one rough and the other soft. It is only within the last few years that I have discriminated

these two murmurs, but during this time my field of clinical observation has been so extensive that I have had abundant opportunities to make the discrimination. With regard to the frequency of the mitral direct murmur, it is by no means so rare as is generally supposed, and as I had thought some years ago. At one time during the past winter, in Bellevue Hospital, I knew of six examples of it, and several also at the Blackwell's Island Hospital. When the auscultator has learned to distinguish it, he will not be long in finding it if he be in the way of seeing a moderate number of cases of disease of the heart. From what has now been said, it is obvious that an important point pertaining to this murmur is, its discrimination from other murmurs. This point will first claim consideration.

In order to comprehend this murmur, it is essential to understand clearly when the mitral direct current of blood takes place. The opportunity of observing the movements of the heart exposed to view in a living animal, conduces greatly to a clear understanding of this point. The mitral direct current is produced by the contraction of the auricles; now, when do the auricles contract? When the movements of the heart are observed, it is seen that the contraction of the auricles immediately precedes the contraction of the ventricles. So close is the connection between the contraction of the auricles and the contraction of the ventricles, that the former appears to merge into the latter; there is no appreciable interval between the two, but the successive movements, although distinct, appear to be continuous. Moreover, it is evident to the eye, and to the touch, that the contractions of the auricles are not so feeble as some seem to suppose. The mitral direct current of blood, therefore, occurs just before the ventricular systole, it continues up to the ventricular systole, and must, of course, instantly cease when the ventricles contract. The contraction of the ventricles causing the first sound of the heart, it follows that the mitral direct current caused by the auricular contractions must take place just before the first sound; that it must continue to the first sound, and that it cannot continue an instant after the first sound.

The mitral direct murmur is produced by the mitral direct current of blood forced by the auricular contractions through a contracted or roughened mitral orifice. Hence, the facts just stated with regard to the current, apply to the murmur. The murmur occurs just before the ventricular systole or the first sound of the heart; it continues up to the occurrence of the first sound, and instantly ceases when the first sound is heard. It is not strictly correct to call this a diastolic murmur; it does not accompany the second or diastolic sound of the heart. The aortic regurgitant is the only true diastolic murmur. The mitral direct is a pre-systolic murmur; this name expresses its proper relation to the heart sounds, and it is the only murmur which does occur in that particular

relation. The time of its occurrence as just explained, and as expressed by the term pre-systolic, is sufficient for its easy recognition when once it is fully comprehended. Although, when this murmur is fully comprehended, and has been repeatedly verified, it is more readily recognized than either of the other murmurs, there is often at first considerable difficulty in determining its existence. Let me endeavour to point out the way in which it may be ascertained. I have already said that by those who overlook this murmur it is generally confounded with the mitral systolic or regurgitant murmur. This is in consequence of its close connection with the first sound, and because it is heard at and near the apex of the heart. Now it is evident that a mitral systolic murmur cannot commence before the ventricular systole. It is equally evident that the ventricular systole and the first sound of the heart are synchronous. It is, therefore, an absurdity to suppose that a mitral systolic or regurgitant murmur can be pre-systolic in the time of its occurrence. This murmur must necessarily accompany and follow the first sound of the heart, as clinical observation has established. We have, then, only to determine that a murmur is pre-systolic, and that it does not accompany the second sound of the heart (*i e*, there is an appreciable interval of time between the second sound and the murmur), to recognize it as a mitral direct murmur. Generally it is sufficiently easy, after a little practice, to perceive that the murmur precedes the sound, but, if there be difficulty or doubt, there is a ready mode of rendering it apparent, this is by placing the finger on the carotid pulse. The carotid pulse is synchronous with the first sound of the heart, or, at least, so nearly synchronous, that there is no appreciable interval of time between them. Placing, then, the finger on the carotid and listening to the murmur at the apex, the murmur is found to occur before the arterial impulse and to cease instantly when the latter is felt.

The mitral direct murmur is to be discriminated from an aortic regurgitant murmur. These two murmurs may be confounded at first, but after a little practice the discrimination is easy. The aortic regurgitant murmur accompanies and follows the second sound of the heart, the mitral direct commences after the second sound. Generally there is a distinctly appreciable interval of time between the second sound and the commencement of the murmur. The aortic regurgitant murmur may be prolonged nearly or quite through the long pause up to the first sound, but the intensity of the murmur diminishes with the prolongation, the murmur being insensibly lost before or when the first sound occurs. The mitral direct murmur on the contrary, always continues up to the first sound, and instead of losing any of its intensity, it becomes more intense, and appears to be abruptly arrested, in its greatest intensity, when the first sound occurs. This is a striking characteristic. The difference in the situation in which two murmurs respectively are heard with their

maximum of intensity, is another point in the discrimination. The aortic regurgitant murmur is generally heard at the base of the heart, and is heard loudest a little below the base near the left margin of the sternum on a level with the third intercostal space. The mitral direct murmur is heard loudest at or a little within the apex, is generally confined within a circumscribed space, not propagated much without the apex and rarely to the base of the heart.

The quality *of the mitral direct murmur is, in many cases, characteristic.* In my work on diseases of the heart I have said that this murmur is generally soft. My experience since that work was written has shown me that this statement is incorrect. The murmur is oftener rough than soft. The roughness is often peculiar. It is a *blubbering* sound, resembling that produced by throwing the lips or the tongue into vibration with the breath in expiration. I suppose that the murmur is caused, in these cases, by the vibration of the mitral curtains, and that the vibration of the lips or tongue by the breath represents the mechanism of the murmur as well as imitates the character of the sound. At one time I supposed this blubbering murmur denoted a particular lesion, viz., adhesion of the mitral curtains at their sides, forming that species of mitral contraction known as the *buttonhole slit*, but I have found this variety of murmur to occur without that lesion, and, in fact, as will be seen presently, when no mitral lesion whatever exists.

A mitral direct murmur may, or may not, be associated with a mitral systolic murmur. Without having analyzed the numerous examples which I have recorded during the last few years, I should say that, while the mitral systolic murmur is much more frequent in its occurrence than the mitral direct, the former, indeed, being the most common of all the murmurs, the mitral direct is observed quite as often without, as with the mitral systolic. But the two frequently coexist, and then the demonstration of the existence of the mitral direct murmur may be made more striking than when it exists alone, provided, as is usually the case, this murmur be rough and the mitral systolic murmur be soft. Listening at or near the apex in a case presenting a blubbering mitral direct and a soft mitral systolic murmur, the former, of course, precedes the latter, and between the two occurs the first sound of the heart, the apex-beat and the carotid pulse. The first sound, the apex beat or the carotid pulse will be found to mark the abrupt ending of the mitral direct, and the beginning of the mitral systolic murmur. The different relations of the two murmurs to the first sound are distinctly perceived in such a case if the observer be prepared to perceive them by a clear comprehension of the subject. And when once the discrimination between the two murmurs has been fairly made, it becomes sufficiently easy, indeed, the mitral direct murmur is then more readily recognized than either of the other murmurs.

The existence of a mitral direct murmur has been theoretically denied on the ground that the auricular contractions are too weak to propel the current of blood with sufficient force to give rise to a sound It is undoubtedly true that, other things being equal, the intensity of a murmur is proportionate to the force of the current, and clinical observation shows that sometimes a murmur is not appreciable when the heart is acting feebly, but becomes distinct when the power of the heart's action is from any cause increased But murmurs do by no means always require for the production a powerful action of the heart, on the contrary, loud murmurs are often found when the heart is acting very feebly For example, I have reported a case in which an aortic direct and an aortic regurgitant murmur were well marked in a patient an hour before death, the patient dying from paralysis of the heart due to distension of the left ventricle Venous murmurs in the neck are often notably loud when, assuredly, the force of the current of blood in these veins is vastly less than the current from the auricles to the ventricles The feebleness of the current in this instance is shown by the slight pressure requisite to interrupt it and arrest the murmur It requires but little force of the expiratory current of air to throw the lips into vibration so as to produce a loud sound Moreover, one has only to see and feel the contractions of the auricle, when the heart is exposed in a living animal (the heart's action being much weakened under these circumstances) to be convinced that the power of these contractions is not so small as some seem to imagine, the blood is driven into the ventricles with considerable force It is hardly necessary to say, however, that *a priori* reasoning with regard to the existence or non-existence of physical signs is not admissible Their existence is a matter to be determined by direct observation Clinical observation shows that a murmur does occur at the precise time when the mitral direct current takes place as shown by observation of the movements of the heart exposed to view in a living animal And clinical observation shows that this murmur is not always feeble, but, on the contrary, is not infrequently notably loud

So much for the reality of the mitral direct murmur and the means of discriminating it from other murmurs It remains to consider another important practical point, viz, the pathological import of this murmur As already stated, it is developed in connection with a contracted mitral orifice, and, so far as my experience goes, especially in connection with contraction caused by adherence of the mitral curtains, forming the *buttonhole slit*, the murmur, then, being due, not to the passage of blood over a roughened surface, but to vibration of the curtains And the sound, as thus produced, is peculiar, resembling the sound which may be produced, in an analogous manner, by causing the lips to vibrate with an expiratory puff The murmur however may be produced by the flowing of the current of blood over a roughened surface, without contraction of the aperture This

is undoubtedly rare As a rule, the force of the mitral direct current is not sufficient to develop a murmur unless there be mitral contraction Is this murmur ever produced without any mitral lesions? One would *a priori* suppose the answer to this question to be in the negative Clinical observation, however, shows that the question is to be answered in the affirmative I have met with two cases in which a well-marked mitral direct murmur existed, and after death in one of the cases no mitral lesions were found, in the other case the lesion was insignificant I will proceed to give an account of these cases, and then endeavour to explain the occurrence of the murmur

Case 1[1] In May, 1860, I examined a patient, aged 56, who had had repeated attacks of palpitation, sense of suffocation, with expectoration of bloody mucus and a feeling of impending dissolution, but without pain, the paroxysms resembling angina, excepting the absence of pain In the intervals between these attacks he was free from palpitation, did not suffer from want of breath on active exercise, and considered himself in good health He had never had rheumatism On examination of the chest, the heart was found to be enlarged, the enlargement being evidently by hypertrophy At the apex was a pre-systolic blubbering murmur, which I then supposed to be characteristic of the buttonhole contraction of the mitral orifice At the base of the heart was an aortic regurgitant murmur, which was diffused over nearly the whole praecordia There was no systolic murmur at the base or apex Three days after this examination the patient was attacked with another paroxysm, and died in a few moments after the attack, sitting in his chair The heart was enlarged, weighing 16½ oz, the walls of the left ventricle measuring four-fifths of an inch The aorta was atheromatous, and dilated so as to render the valvular segments evidently insufficient The mitral valve presented nothing abnormal, save a few small vegetations at the base of the curtains, as seen from the auricular aspect of the orifice

In this case it is assumed that the mitral direct murmur, which was loud and of the blubbering character, was not due to the minute vegetations which were found after death There was no mitral contraction The mitral valve was unimpaired, so that the murmur could not have been due to mitral regurgitation

Case 2[2] In February, 1861, I was requested to determine the murmur in a case at the Charity Hospital, New Orleans I found an aortic direct and an aortic regurgitant murmur, both murmurs being well marked There was also a distinct pre-systolic murmur within the apex, having the blubbering character On examination after death, the aorta was dilated and roughened with atheroma and calcareous deposit The aortic segments were contracted, and evidently insufficient The mitral curtains presented no lesions, the mitral orifice was neither contracted nor dilated,

[1] Private Records vol x p 713
[2] Ibid, vol xi p 241

and the valve was evidently sufficient. The heart was considerably enlarged, weighing 17½ oz., and the walls of the left ventricle were an inch in thickness.

In the second, as in the first of the foregoing cases, it is evident that a mitral systolic murmur was not mistaken for a mitral direct murmur, for in both cases, the conditions for a mitral systolic murmur were not present. In both cases the mitral direct murmur was loud and had that character of sound which I suppose to be due to vibration of the mitral curtains. In both cases, it will be observed, an aortic regurgitant murmur existed, and aortic insufficiency was found to exist post mortem. How is the occurrence of the mitral direct murmur in these cases to be explained? I shall give an explanation which is to my mind satisfactory.

The explanation involves a point connected with the physiological action of the auricular valves. Experiments show that when the ventricles are filled with a liquid, the valvular curtains are floated away from the ventricular sides, approximating to each other and tending to closure of the auricular orifice. In fact, as first shown by Drs. Baumgarten and Hamernik, of Germany, a forcible injection of liquid into the left ventricle through the auricular opening will cause a complete closure of this opening by the coaptation of the mitral curtains, so that these authors contend that the natural closure of the auricular orifices is effected, not by contraction of the ventricles, but by the forcible current of blood propelled into the ventricles by the auricles. However this may be, that the mitral curtains are floated out and brought into apposition to each other by simply distending the ventricular cavity with liquid, is a fact sufficiently established and easily verified. Now in cases of considerable aortic insufficiency, the left ventricle is rapidly filled with blood flowing back from the aorta as well as from the auricle, before the auricular contraction takes place. The distension of the ventricle is such that the mitral curtains are brought into coaptation, and when the auricular contraction takes place the mitral direct current passing between the curtains throws them into vibration and gives rise to the characteristic blubbering murmur. The physical condition is in effect analogous to contraction of the mitral orifice from an adhesion of the curtains at their sides, the latter condition, as clinical observation abundantly proves, giving rise to a mitral direct murmur of a similar character.

A mitral direct murmur, then, may exist without mitral contraction and without any mitral lesions, provided there be aortic lesions involving considerable aortic regurgitation. This murmur by no means accompanies aortic regurgitant lesions as a rule, we meet with an aortic regurgitant murmur frequently when not accompanied by the mitral direct murmur. The circumstances which may be required to develop, functionally, the latter murmur, in addition to the amount of aortic regurgitation, remain to be ascertained. Probably enlargement of the left ventricle is one

condition The practical conclusion to be drawn from the two cases which have been given is, that a mitral direct murmur in a case presenting an aortic regurgitant murmur and cardiac enlargement, is not positive proof of the existence of mitral contraction or of any mitral lesions The coexistence of a murmur denoting mitral regurgitation, in such a case, should be considered as rendering it probable that the mitral direct murmur is due to contraction or other lesions, and not functional

Dr Gairdner, in a recent article already referred to, proposes a change of name for the mitral direct murmur He proposes to call it an auricular systolic murmur Inasmuch as the murmur is produced by the systole of the left auricle, this name is significant And the usual name is open to this criticism, viz , it is not produced by the whole of the mitral direct current, but only that part of the current which is caused by the contraction or systole of the auricle From the situation of the auricles as regards the ventricles, the former being placed above the latter, and the free communication by means of the auriculo-ventricular openings, the blood must begin to flow from the auricles into the ventricles the instant the ventricular contractions cease During the first part of the long pause or interval of silence, *i e*, the period after the second sound and before the subsequent first sound of the heart, the blood flows from the auricles into the ventricles simply in obedience to gravitation It is not ascertained that this part of the current ever gives rise to a murmur If it does, the murmur would follow immediately the second sound, or when an aortic regurgitant murmur occurs I have conjectured that such a mitral direct murmur may occur, and that it is confounded with an aortic regurgitant murmur This conjecture is based on cases in which an apparent aortic regurgitant murmur existed, and the aortic valves seemed to be nearly or quite sufficient on examination after death However this may be, the mitral direct current giving rise to the murmur which has been considered in this article, is not the current which immediately follows the second sound, and is due to gravitation alone, but it is the current immediately preceding the ventricular systole, and due to the systole of the auricle Hence, as it seems to me, the name proposed by Dr Gairdner, being more specific and accurate, is to be preferred to that in common use

1867

PIERRE CARL ÉDOUARD POTAIN

DESCRIPTION OF THE MOVEMENTS AND THE MURMURS OF THE JUGULAR VEINS

PIERRE CARL E POTAIN

(Courtesy Charles C Thomas)

PIERRE CARL EDOUARD POTAIN

(1825-1901)

PIERRE CARL EDOUARD POTAIN, who was born in Paris on July 19, 1825, came from a long line of physicians. indeed, one of his ancestors is known to have practiced surgery in 1662. Potain, himself, used to say, "Without a doubt, there must have been some barber at the origin [of the family], but it was in a time too remote to be found now."

Potain's father was postmaster of Saint-Germain and set his heart on his son's returning to the family tradition of medicine. When Potain was very young, his father took him, at the close of day, to the forest of Saint-Germain and taught him all that he knew of the natural sciences, grammar and literature. He learned the German language from his mother.

Potain could not afford a college education, but he studied by himself and soon was able to pass the examinations at the University of Paris, where he received the degree of Bachelor of Arts. His own tastes inclined him to science and mechanics, but urged by his father, he began the study of medicine, and in 1848 passed the examinations which permitted him to serve an internship at the hospitals in Paris. In 1849, while at the famous Salpêtriere Hospital, he suffered an attack of cholera, but soon recovered from it. While convalescing at Metz he suffered a second attack of cholera, but managed to survive it.

After he received the degree of Doctor of Medicine in 1853 from the University of Paris he decided to accept a position as assistant to Jules Gabriel François Baillarger (1806-1891) at the asylum for the insane at Ivry. In 1856 he returned to Paris, where he worked under Jean-Baptiste Bouillaud (1796-1881), who was the first to describe "gallop-rhythm." Potain became chief of Bouillaud's clinic and later served on the staffs of the Hôpital Sainte-Antoine and the Hôpital Necker. In 1859 he became an assistant professor at the medical school of the University of Paris. In 1861 he took the competitive examination and was named physician of the Hospitals of Paris and associate professor of the Faculty of Medicine of Paris.

In 1870, during the war, Potain was asked to take charge of an ambulance company, but he decided, instead, to enlist in the regular army as a common "carabinier." When not on duty he returned to the hospitals to care for the wounded. In 1876 he was appointed professor of pathology at the University of Paris. Later that year he was transferred to the chair of clinical medicine. In 1882 he became associated with the Charity Hospital in Paris, where he remained until the age of retirement.

In 1883 Potain was elected to the Académie de Médecine, and in 1893, to the Académie des Sciences, the Institute of France. He was elected a commander of the Legion of Honor.

Potain numbered among his patients Henri C. F. M. Dieudonné, Comte de Chambord (1820-1883). On one occasion when he received a call from the count, who was the Pretender to the throne of France, he sent his friend, Edmé Félix Alfred Vulpian (1826-1887), to see the count while he stayed to comfort an ill friend, Dr. Parrot, for whom he knew very well he could do nothing.

In 1900 Potain reached the age of retirement He was sorry to leave Louis Henri Vaquez (1860-1936), his former pupil and Louis Joseph Teissier (1851-1926), his assistant, went to wish him a Happy New Year on January 1, 1901, and told him to come back to do research To this Potain replied "No, you see, it is well over, when the function is through, the organ must disappear"

He died suddenly within the week, and was buried on January 8, 1901

Potain realized the importance of recording the heart beats in connection with the venous pulsations and the graph of the pulse, and he devised an instrument with which to make these recordings possible

His chief contributions to medicine included (1) making precise the relation ship of tricuspid regurgitation and circulatory disturbances, (2) description of the pulsation of the liver, (3) distinguishing between the different varieties of gallop rhythm, (4) explanation of the mechanism of the apex beat, and (5) recognition of the "bruit de tabourka,"[1] the second heart sound, which occurs in the presence of syphilitic aortitis

Potain long had been interested in the measurement of arterial pressure, which Karl Vierordt (1818-1884) had advocated and for which Étienne Jules Marey (1830 1904) had provided the means of evaluation Marey's instrument, however, was neither precise nor practical According to Vaquez, Potain learned "with joy" of Von Basch's invention of sphygmomanometry Von Basch's instrument was a cumbersome affair, however, and Potain improved upon it and made its use practicable

Potain's development of the portable sphygmomanometer led him to new discoveries It permitted him to demonstrate the reality of hypertension in Bright's disease, as suspected by Traube It also enabled the detection of hypertension in other pathologic conditions to be made Potain, moreover, demonstrated that hypertension and not the impaired function of the kidney was responsible for the almost constant cardiac hypertrophy found in renal sclerosis

The mechanical genius of Potain is also shown in many other ways He perfected an apparatus to count red blood cells (Malassez hematimeter) In studying the congestive form of pulmonary diseases, his intern Georges Dieulafoy had chanced on the discovery of pleural aspiration Potain helped him perfect an aspirator with a vacuum apparatus (Potain's apparatus), which is still in use By means of this new apparatus he was destined to add further to his renown For a patient who had extreme dyspnea, Potain performed thoracentesis and completely withdrew the fluid by means of his vacuum apparatus With another apparatus, which he had constructed for the occasion, he replaced the fluid by air progressively introduced to avoid undue expansion of the lung He used the same procedure for twenty patients, obtaining equally favorable results He can, therefore, be said to have perfected, and he later reported the first indisputable facts extant on the favorable influence of collapse therapy in the evolution of the treatment of tuberculosis

Potain was a discreet writer and he worked for a long time before he announced his discoveries, feeling that he might be premature in his judgment When he did express his thoughts in writing, however, they were generally in the form of short memoirs, and were models of exposition in clearness and style One of these short memoirs, "Théorie du Bruit de Galop" (1885), it is our privilege to reproduce in translation[2] We are also presenting to our readers a translation of his classic description of the bruits of the jugular veins, one of his earliest works, published in 1867

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[1]Gibson G A *Diseases of the Heart and Aorta,* New York, 1898 The Macmillan Company p 827

[2]See page 652

ON THE MOVEMENTS AND SOUNDS THAT TAKE PLACE IN THE JUGULAR VEINS*

By

DOCTOR POTAIN

Associate Professor of the Faculty of Medicine, Physician to Necker Hospital

Gentlemen

You have heard in our recent meetings two very important papers concerning the murmurs of vessels The one of M Parrot and the other of M Peter Both of these gentlemen have questioned the diagnostic value which is generally granted to vascular sounds in the diagnostics of anemia, and both are of the opinion that it is necessary to seek the immediate cause of these phenomena, whatever additional may be the share of the state of the blood itself Each stated further that he believed he was able to specify this cause, M Parrot maintained that it is due to insufficiency of the valves of the jugular vein, M Peter, to a spasm of the venous or arterial wall

I also desire to say a word on this subject, not so much to dispute the opinions of my two colleagues as to present, in turn, the results of researches concerning this question which I have carried out over a period of several years Until now I have not considered my study far enough developed to present to you, and now I do not believe by any means that I have obtained a definite solution But since the question of vascular sounds has been raised among us, I do not wish to withhold them any longer

The results at which I have arrived are in agreement in some respects with the ideas presented by my colleagues, in others they do not agree Like these gentlemen, I believe that vascular murmurs are not pathognomonic of anemia As with them it has appeared to me that this murmur could exist and be found most intense in persons not at all anemic whereas it is entirely absent in persons who are obviously anemic to a high degree Exceptions were known and pointed out by M Bouillaud in his first studies, but they are more numerous, without doubt, than is generally believed I admit with my colleagues, following the opinion of M Chauveau, that the veins are the site, not only of continuous murmurs but also of the majority of intermittent murmurs which are heard over the neck

I am in complete disagreement with each of them as to the further specification of the mechanism of the venous murmurs and as to their clinical value Because, on the one hand, my observations do not permit

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me to admit that the ordinary cause of these murmurs is concerned with a venous reflux, nor with a spasm of the great vessels, on the other hand they lead me to believe that the variable proportion of blood corpuscles cannot but have a direct and markedly appreciable influence on the production of these murmurs, and, furthermore, that certain types of these abnormal sounds may moreover be considered as characteristics of anemia.

For the remainder, the points of this question which our colleagues have taken up are concerned at the same time with the mechanism and with the clinical value of the vascular murmurs, we shall return then to these two aspects of the problem. Let us see at first that which concerns the mechanism.

I. OF THE MECHANISM OF VENOUS VASCULAR MURMURS WHICH ARE HEARD IN THE NECK

When one observes carefully the portion of the supraclavicular region which the jugular veins and the carotid artery cross, one may frequently note three distinct phenomena through which are revealed the motion by which the blood is quickened in the vessels: (1) visible oscillations, (2) a thrill sensible to the finger, (3) normal and abnormal sounds revealed by auscultation. Since an analysis of the movements and the peculiarities which the purring fremitus shows may throw some light on the mechanism of the murmurs, I have applied myself to study them with care in a large number of individuals, and here is what I have found concerning this subject in repeated observations.

(1) The visible oscillations in this region consist of a series of filling and collapses, sometimes prominent and easy to recognize. In some persons they are appreciable only in the lowest part, that is, nearest the clavicle, in others they are seen distinctly over a large portion of the vessels, exceptionally, one may follow them over the course of the external jugular.

Because of their unequal amplitude, they are not always equally obvious and sometimes one or more are completely lacking, but when they are well marked, when no affection of the heart alters the rhythm and when the pulse is not too fast, their study is generally quite easy. There is found then, aside from the slow oscillations caused by the respiratory movements and simultaneous with them, the following sequence of movements which is repeated with constant and perfect regularity: at first a slow elevation, then two quick elevations, finally two deep depressions, after which the series begins again. Now each series of this kind corresponds to a cardiac cycle.

These impulses sometimes have such force and amplitude that at first it might be believed that they represent pulsations of the carotid artery or of the subclavian. But after a little attention one is soon convinced that they actually take place in the internal jugular. This is proved thus

In the first place they are of a vague and diffuse nature, totally opposite to the idea of an arterial pulsation, and yet apparently, however extensive they may be, they are felt with difficulty by the finger which perceives with intensity pulsations of very slight extent either at the same level and nearby, or a little higher along the course of the carotid, in the second place, their rhythm does not resemble in any respect that of the pulsations of the carotid, the proof of which you shall see now in the totally different form of the tracings shown by the sphygmograph, in the third place and finally, a light pressure, suitably applied to the lower portion of the neck can impede them or suppress them entirely, while the pulsations of the carotid persist with all of their intensity

These movements verified and their site established, it remains to seek an interpretation It appeared to me that the most certain means would be to determine exactly their relationships with the different portions of the cardiac cycle I studied them then in this relationship by combining palpation or auscultation of the precordial region with inspection of the cervical pulsations, and, in this manner, I arrived at these results The first of the two sudden elevations immediately precedes ventricular systole, while the second coincides nearly exactly with it, the first depression takes place during the short silence, and the second, immediately after the second heart sound, that is, at the time of ventricular diastole, finally, the slow elevation which initiates the series, occurs in the middle of the long silence, that is, during cardiac rest

This method of study permitted the clear establishment of the relation between the jugular pulsations and the movements of the heart, and I have found that the two elevations observed correspond to the successive contractions of the auricle and ventricle, and the two depressions to the diastoles of these cavities In fact, there can be little question but that the second quick elevation appears to follow the systolic impact and first heart sound immediately, and to coincide exactly with the pulse of the large arteries As to the first, which precedes it, occurring before the contraction of the ventricle, it certainly cannot have for a cause an action which it precedes, it must then be attributed to the systole of the auricle Finally, the two depressions appeared to me to occur at the precise moments which we know are occupied by ventricular and auricular diastole, and their appearance, at this moment, is explained without difficulty, because it is entirely natural to see the veins collapse suddenly at the moment when the blood that they contain is propelled toward the cardiac cavities, which pass suddenly from a state of contraction to one of relaxation

But the comparative study of venous pulsations and movements of the heart, even though these movements are of slow frequency, is a very delicate thing, because it requires the comparison of impressions received by two different senses, which is always difficult Moreover, I

have become accustomed by patient observation to understand clearly the sequence and relationships of these somewhat complicated movements, but I could not expect to demonstrate them readily. Furthermore as soon as the heart beat is accelerated a little, this study becomes impossible and the relationships are appreciable with great difficulty. I decided then, in order to study them with more precision and in a more demonstrable manner, to use the valuable sphygmographic apparatus which we owe to our colleague, Marey.

The idea of obtaining a graphic representation of the jugular pulsation is not at all new. In Germany, Bamberger, Geigel, and Friedreich have already employed it in the study of exaggerated pulsations seen in certain cardiac diseases, and Friedreich has shown that one may obtain a similar tracing in the absence of a pathologic venous pulse. But these observers were content to apply the sphygmograph designed by Marey for recording the radial pulse to the region occupied by that vessel. There resulted, in the first place, many difficulties in the application of the instrument, in the second place, a complete impossibility of determining then with any certainty the significance of the various portions of the tracing and their relationship to the different motions of the heart. But, this last point being exactly that which I sought to establish, I had to approach differently.

I employed the addition which Marey has made to his instrument to transform it into a cardiograph. I recorded the jugular pulsations with the help of a small glass funnel which acted as a stethoscope and transmitted the impulses received to the tambour of the instrument through an India-rubber tube. Simultaneously I applied the same sphygmograph to the radial artery to which were transmitted the pulsations of the jugular. Then I arranged things in such a manner that the two levers, that of the jugular and that of the radial, would write their tracings at the same time, on the same paper, and one above the other. Not content with this, I placed the funnel, or another instrument better arranged, over the precordium and thus recorded the impulses of the heart simultaneously with those of the radial pulse. Finally, for more certainty, while the pulsations of the heart were being recorded, I placed the sphygmograph directly over the carotid in cases in which this artery was easily accessible. It only remained to compare these different tracings and to superimpose them with care by means of a very precise method, but which would be too long to discuss here, to see coincidences established in some manner of themselves and in the most strict fashion which can be imagined.

Here, Gentlemen, is an example which has been enlarged[1] to show to you.

You may see at once in the first tracing of this figure (I) the exact reproduction of the movements which I just now described to you after

[1]The figure placed here is a reproduction of the tracing in its original size.

simple inspection a progressive rise (A), two short elevations of little extent (B) (C), then two deep depressions (D) (E) You will note further that the same movements are reproduced at each cardiac cycle, modified only by the respiratory oscillations which are added and which cause some changes in the line as a whole without ever suppressing any of the details mentioned In the same figure are reproduced, with the help of the procedures which I have outlined to you and with the coincidences marked exactly by vertical lines, the radial pulse (II), the carotid pulse (III), the impulse of the apex of the heart (IV) I believe that on comparing these four tracings one may determine the precise significance of each part of the first, as you may judge

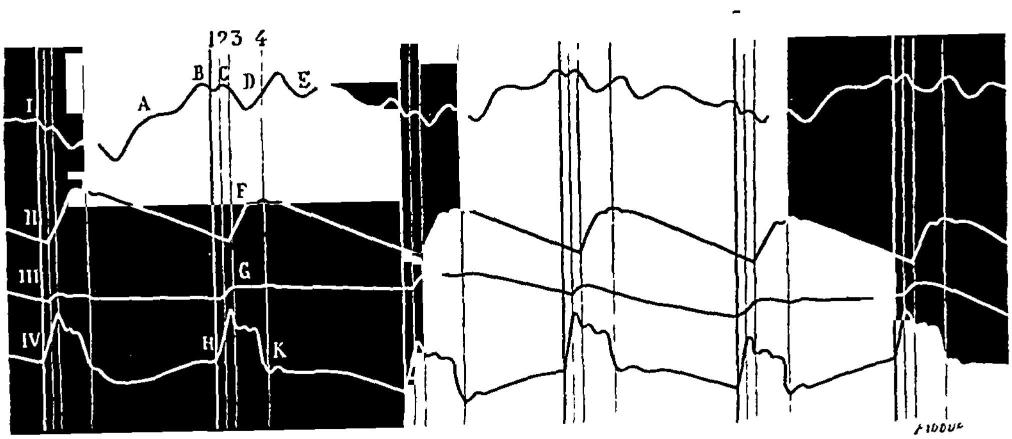


Fig 1

I Pulsations of the jugular vein
II Pulsations of the radial pulse
III Pulsations of the carotid
IV Pulsations of the apex of the heart

A Progressive repletion of the vein
B Elevation caused by *contraction of the auricle*
C Elevation caused by *contraction of the ventricle*
D Depression produced by *diastole of the auricle*
E Depression produced by *diastole of the ventricle*
H Beginning of ventricular contraction
K End of ventricular contraction, occlusion of the semilunar valves, beginning of diastole

1 Line of beginning of ventricular systole
2 Line of the carotid pulse
3 Line of the radial pulse
4 Line of *ventricular* diastole

To show at first that the tracing (I) represents the pulsations of the jugular and not those of the carotid, it will suffice, in the absence of other proof, to compare it with that obtained when the instrument is placed over the carotid itself (III), and to note that the latter does not show any details which the former shows, and does not resemble it in any manner, further, to observe that the movement (B) takes place at a time when no impulse has been produced in the arterial system, because the ventricle (H) has not yet begun to contract

If we now analyse the jugular tracing, we shall see there clearly what follows (1) the first quick elevation (B) precedes the radial pulse considerably (F), it precedes the carotid pulse (G) also, although by somewhat less and most remarkable yet, the beginning of ventricular systole (H), consequently it could not be brought about by this systole which it precedes On the contrary it is produced at the precise instant where physiology tells us that the contraction of the auricle takes place, and since there is no motion of the ventricle at this moment, it must be attributed necessarily to the contraction of the auricle It is thus readily explained by the light reflux which auricular systole causes in veins near the thorax, (2) the prominence (C) immediately follows ventricular systole (H), and coincides exactly with the carotid pulse (G) Like the latter, it is the result then of ventricular systole, perhaps the systolic movement is transmitted directly to the venous system at the moment when closure of the tricuspid valve takes place, perhaps it is transmitted indirectly by the compression which the arterial trunks in diastole certainly exert on the venous trunks which they are near, perhaps, finally, it results from these two modes of action combined, (3) the first depression (D) takes place in the interval between the vertical lines 3 and 4 which indicate, the one, the beginning of the radial pulse (F), the other, the moment of ventricular diastole (K), that is, it is produced during the time when the ventricle contracts (HK), and in the moment when the carotid artery is in complete diastole (G) Consequently, it cannot have its cause either in the contraction of the artery or in the relaxation of the ventricle But it corresponds precisely to the time when diastole of the auricle takes place, it should then result from the rapid flow of venous blood into this relaxed cavity, an afflux which immediately empties the veins near the thorax, (4) finally, the second depression (E) occurs a little after ventricular diastole (K), and it cannot be very well explained except by the new impulse which this diastole brings to the blood contained in the auricle and which is transmitted to the veins of the neck

The tracing which we have analysed was taken on a woman in childbed in l'Hôpital Necker, in whom the pulse, very much slowed, beat only 40 times each minute I have chosen this case to present to you first because this extreme slowness is especially helpful in the study of the coincidences which I wished to establish Those which I have obtained in other cases of the same kind, are entirely identical, but, since you may think that the special state of the circulation in women in childbed might give rise to pulsations in the vessels which would not exist in the absence of this state, here on a second plate (Fig 2) is a tracing taken from a man in whom the pulse, slowed by icterus, beat 46 times per minute

You may see at once that the form of the tracing is entirely similar to that of the preceding one, that is, the elevations and depressions take

place in the same manner and in the same order, you note, further, that the coincidences are the same Indeed, you see, as in the preceding one, at A the slow rise which corresponds to the absolute rest of the heart and which indicates the slow filling of the vein, at B the sudden elevation which precedes ventricular systole (H) and must be attributed to the contraction of the auricle, at C, the elevation coincides exactly with the systole of the ventricle, at D, the first depression that takes place during this systole and which represents the diastole of the auricle, finally, at E, the second depression which corresponds exactly with ventricular dilatation (K)

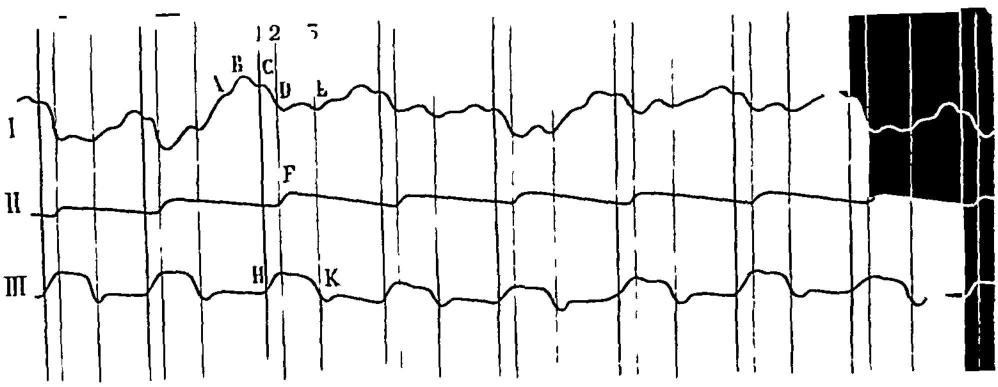


Fig 2

I Jugular vein
II Radial pulse
III Apex of the heart

A Progressive repletion of the vein
B Contraction of the auricle
C Contraction of the ventricle
D Dilatation of the auricle
E Dilatation of the ventricle

1 Line of ventricular systole
2 Line of the radial pulse
3 Line of ventricular diastole

Finally, it is not necessary to have a pathologic state to show the pulsations of the jugular vein with this completeness and with all of the details which I have shown you in the preceding tracings I show you, in proof of this, one which I have taken on myself (Fig 3)

The elements of the curve are a little closer together, because my pulse is more rapid than that of the patients on whom I made the preceding tracings but they have the same form, they exhibit the same order and are easily recognizable Thus one may note at A, the slow elevation produced by the progressive filling of the vein, at B, the quick elevation due to the systole of the auricle, at C, that of ventricular systole, at D, the depression of auricular aspiration, at E, that of ventricular aspiration, that is to say precisely all that which we have discovered in analysing tracings shown by the jugulars of women in childbed or of icteric persons with slow pulse

Let us see now what are the relations of these movements to thrills and murmurs

(2) The *thrill,* which is felt on placing the finger lightly above the clavicle over the course of the vessels of the neck, is sometimes continuous and frequently intermittent, it is this last case which interests us above all here When one observes it attentively and especially on himself, one finds that an energetic and sustained inspiration reinforces it if it is weak, or *tends to make it continuous if it is intermittent, that a forced expira*tion stops it immediately, that, sometimes, a light pressure exerted above the point of exploration can make it appear or reinforce it, while a stronger pressure extinguishes it completely Proofs positive, if they are considered with attention, that we are concerned with a venous phenomenon and that this thrill does not arise at all from an artery Sometimes also, it is possible to determine in a manner perfectly evident, that the intermittent thrill appears at the precise moment when the vein collapses, and that it is single or double, the same as the collapse to which it corresponds Now, it is evident of itself and super-abundantly

Fig 3

Tracing of the pulsation of my jugular

A Progressive repletion of the vein
B Systole of the auricle
C Systole of the ventricle
D Auricular diastole
E Ventricular diastole

proven, I believe, by the facts presented above, that the depression of the region occupied by the internal jugular corresponds to a flow of blood toward the cavity of the chest, with the result at this moment, that the flow in this vein is accelerated There is then good reason to believe that the acceleration of the centripetal flow is the true cause of the reinforcement which the thrill shows at this moment Also I venture to say (if the facts which I have presented have been, as I believe, well observed) that there could not be any other rational interpretation for this fact

(3) The *murmur* coincides with the thrill, is seen in the same circumstances, is present at the same point and with the same varieties of rhythm Why can we not recognize the same cause and the same mechanism for these two phenomena? Why should we doubt that the interpretation applicable to the one should be equally well applicable to the other? The resemblance is at least very great However, this proof by induction does

not suffice here, I wanted a more direct proof, and here is the very simple artifice which gave it to me

I have placed, as you see here, a small funnel of glass with an opening about the size of the bell of a stethoscope at one end of an India-rubber tube, the other end of which was introduced into the ear I applied this flexible and transparent kind of stethoscope to the region of the vessels of the neck, and in this manner I could hear the sounds distinctly, while I had the eye fixed on the region, in fact on the same point over which I listened Then, to make the movements of this region more perceptible, I fixed a little piece of colored paper, bent at a right angle, with a little water to the skin covered by the transparent bell of my stethoscope This paper, having the function of a mobile arm, or if you wish, of a sphygmoscope, considerably augmented the apparent amplitude of the movements and made them easy to observe

In observing in this manner, I have always seen and have always proven for the students, as well as for everyone else who has wished to lend himself, that *the murmur takes place at the precise moment when the skin sinks within the area of the stethoscope,* that it is double when there is a double depression and that, on the other hand, it ceases at the instant when the region rises Is it not a proof at once demonstrative and quite simple, that the murmur is not produced at the moment of diastole of the artery and that it is not arterial, further that it cannot be attributed to a venous reflux, but, on the contrary, that it takes place at the same instant in which the vein is twice under the influence of aspiration by the cardiac cavities?

Further, if in auscultation of the vessels of the neck one listens to the heart sounds, which are frequently transmitted distinctly into this region, one may determine without difficulty that the intermittent venous murmur of which we speak here is always produced, not with the first sound but with the second, that is to say that it corresponds, not to auricular and ventricular systole, but to the diastole of these cavities, in such a way that, when it is double, one precedes the second sound and the other follows it immediately Now, the reinforcements of the continuous sound behave in exactly the same manner

On the contrary, arterial sounds which are also heard in this region are characterized by the fact that they coincide exactly with the diastole of the artery and they follow the first heart sound immediately They are distinguished also from the others by a different quality of sound, by a special quickness in their onset, and by the very different influence which compression or the efforts of inspiration and expiration have on them, finally by the impossibility of transforming them into continuous murmurs

Thus it comes about that one may distinguish three types of murmurs in the neck continuous ones, which arise in the veins as many proofs show, intermittent venous ones, single or double, which correspond to the second portion of the cardiac cycle and coincide with the depressions in the

cervical region, that is to say with diastole of the auricles and of the ventricle, intermittent arterial sounds which are produced in the first portion and accompany exactly the impulse of the artery.

You see, Gentlemen, when one studies with care the murmur which is of particular interest here, that is to say the venous murmur of the jugulars, one finds, in its intermittences or in its reinforcements, only one possible interpretation: that each gust of the murmur or each reinforcement results from an acceleration of the venous flow brought about by diastole of the auricle or of the ventricle.

The murmur, as everyone knows, passes easily from intermittence to continuity under the influence of various causes, for example, slight changes in the degree of pressure exerted over the vessels. And this is readily understood. In fact by slightly increasing the pressure of the stethoscope, the blood, retained and accumulated in the vein above the obstacle, soon acquires a tension so strong that it then passes by this obstacle with a continuous movement, without moreover being influenced (by the lower pressure) in any appreciable manner, without the variations which the lower pressure goes through which is present below.

Nevertheless, independent of external modifications of pressure, the murmur is, in the same individual or in different individuals, sometimes continuous, sometimes single intermittent, sometimes double intermittent, or continuous with reinforcement, and it is surprising that a phenomenon, the rhythm of which is regulated by causes working at all times and everywhere, should nevertheless be so variable. The auricle and ventricle always contract and dilate, why does not the murmur always have a double intermittent rhythm, which is the consequence of these movements? Why? Because the aspirations and refluxes or retardations which originate in these cavities are not the only influences which the circulation of blood in the veins obeys, because there are others which operate in a more or less continuous manner, such as: the thoracic aspiration which is exerted on the large venous trunks contained in the chest, the state of the capillary circulation, the tension of the soft parts which surround the vein, the pressure which they exert, certain arrangements of the vein itself. And, according to whether some or others predominate, it is conceivable why there should be a tendency to intermittence or continuity. But these influences are not always easy to determine when one comes to particular cases. Thus, not possessing precise data on this point, I shall not speak further. I am satisfied, for the present, having shown you, to rest on the proof which I believe positive, of the cause to which should be attributed the frequently intermittent rhythm of murmurs heard over the jugular veins.

After all, this method of interpreting intermittent murmurs of the neck is not exactly a novelty. In his paper of 1858, M. Chaveau already pointed out the influence which ventricular aspiration exerts on the flow of blood in the jugulars. But this able investigator doubted that diastole of the auricle

also had any action of this type Moreover his demonstrations did not appear sufficient beyond doubt, since they seem not to have carried personal conviction This is the reason that I believed it necessary to return again to this subject and that I wished to study it anew

The facts which I have presented to you are not, as you know, Gentlemen, in accordance with the theory which has been presented to you by M Parrot My colleague and friend believes, as you know, that the intermittent murmur or the reinforcements of the continuous murmur are the results of a reflux brought about in the jugular veins either by the contractions of the auricle, or by those of the ventricles, when there is insufficiency of the tricuspid valve As to the continuous form, it results, according to him, from a succession of refluxes, and of affluxes exaggerated by the preceding refluxes

Suitably to apply to observed facts, this new interpretation is faulty first, in those cases in which it assumes an insufficiency of the venous valve, the murmur, supposedly produced by the contraction of the auricle, should immediately precede ventricular systole and, in those in which it further concedes tricuspid insufficiency, it should coincide exactly with this systole, secondly, it is also faulty, where the rhythm is double and intermittent, in that the first of the two murmurs should coincide precisely with an elevation of the region to which the stethoscope is applied and the second with a depression But this never takes place On the contrary, I have always seen, that when there are two sounds, the first follows the first valvular sound after an interval, and the second follows the second heart sound immediately, when, on the contrary, the sound is single, I have always heard it begin either immediately before or immediately after the second heart sound, never after, immediately before or exactly with the first Finally, when the circulation was slow enough to permit a good analysis of these phenomena, I have always seen the single or double sound accompany the single or double depression of the region

To the thesis which I adopt, M Parrot opposes the difficulty of conceiving that a sufficiently continuous movement of blood could exist in the jugular vein to give the perfect continuity to the murmur which is sometimes found, he believes that each systole of the auricle or of the ventricle should necessarily bring about a perceptible interruption in the flow

That would surely be inevitable if the jugular opened directly into the auricle, but this is not the case Between this vein and the heart are interposed the great venous trunks inclosed within the thorax and constantly subject to thoracic aspiration, now these venous trunks, having the function of reservoirs and of a source of constant supply, in certain circumstances may weaken or cause the disappearance of the effect of the reflux which results from cardiac contractions Moreover, as M Monneret formerly observed, it should be even more difficult to conceive

of a perfectly continuous murmur produced by a succession of flux and reflux Whatever might be the rapidity with which these alternating movements might succeed one another, there must always be a dead point, as is said in mechanics, that is to say a moment of repose, and no matter how short you wish to assume, the ear will always be able to grasp it I have tried in inert tubes and in the jugulars of cadavers artificially to produce a continuous sound by the most rapid possible succession of two currents in opposite directions, never have I succeeded Further, it has been demonstrated experimentally, as you may now see for yourselves, that a murmur differs in quality and intensity according to whether one listens at a point above or below the point at which the murmur originates One should always observe in a murmur produced in this fashion an alternate change of quality and intensity That is to say that truly uniform continuous murmurs absolutely resist the method of interpretation proposed by our colleague Further, inspiration and expiration which influence the intensity of murmurs in such an obvious manner by modifying the blood flow, according to this hypothesis should influence unequally the two parts of the continuous murmur Expiration should reinforce the murmur of reflux and weaken that of afflux toward the chest, while inspiration should act in an opposite manner, but this is not the case Inspiration reinforces the murmur in a uniform manner, expiration weakens it in the same manner

Thus, then, in addition to the fact that the theory of my colleague is in formal opposition to facts which I believe are well established, it is of itself open to serious objections and it is for that that it is not possible for me to accept it

The theory of arterial or venous spasm which M Peter is concerned with reviving, undoubtedly is not in as evident opposition to the previously mentioned facts, nevertheless, in its turn it raises objections no less serious You have seen, indeed, that it rests chiefly (on the following) first, that without it one may not explain how the vascular murmurs show variations so frequent, so sudden, so completely independent of changes which might take place in the composition of the blood, second, on the contractility of vascular walls which is generally admitted today

It is incontestable that one cannot establish any precise and exact relationship between these murmurs and the state of the blood, and that these abnormal sounds frequently show an extraordinary variability, on the other hand, venous contractility is undoubtedly a fact completely demonstrated in physiology But you shall see, Gentlemen, in what remains for me to say, that these apparent anomalies may have explanations entirely different from that which has been given by Laennec, and, in my opinion, better founded

Moreover, since nothing can be shown directly as to whether or not the venous wall is in a particular state of contraction in individuals in whom

the murmur is found in the neck, this contraction always remains in the state of a pure hypothesis Consider further that this hypothesis is not successful and that it is very poorly applicable to facts which it is supposed to explain

In fact let us admit that a contraction may take place in the jugular vein of a subject being examined, this contraction either extends the full length of the vessel or it is limited to a circumscribed portion In the first case, it is probable that it would make the murmur disappear, if one existed previously, because its effect would be to retard the flow of blood and to diminish the quantity which would pass through the vessel In the second, after all, the pressure would not do anything more than the pressure of the stethoscope can do and to a degree which the stethoscope can always attain since the pressure of the stethoscope can always be carried to the point of absolute suppression of the murmur by effacing the lumen of the vessel If, then, venous contraction is the only cause of murmurs, since the pressure of the instrument can do the same as venous contraction, the stethoscope should be able to produce a murmur in every case and there would be no question as to why certain people do not show it at all although they may be quite anemic

This theory of vascular spasm is, then, a hypothesis without possible demonstration, which is entirely unnecessary to explain the facts and which gives a poor explanation when one attempts to apply it to them, thus I believe that it should be absolutely rejected

II OF THE CLINICAL VALUE OF VASCULAR MURMURS

In what concerns the clinical value of vascular murmurs, that is to say, their relationship to anemia, I agree with the opinion of my two colleagues, without, however, adopting it entirely M Parrot distinguishes two cases in one, the murmur has absolutely nothing to do with the state of the blood, its cause is insufficiency of the valves of the jugular vein, a purely accidental insufficiency In the other it is not without some relationship with anemia, but this is a distant and indirect relationship, since it results from insufficiency of the tricuspid valve, an insufficiency which should be rather frequent in persons affected by anemia As to M Peter, the relation of the murmur to anemia is always indirect, distant and therefore contingent The immediate cause of the murmur is a spasm of the vein which the poverty of the blood promotes, as it promotes all spasmodic states—As for me, I continue to believe that in the hydremic state there is a condition much more directly favorable to the production of murmurs, and that this state, carried to a certain degree, can produce murmurs without any vital act, without any modification of the caliber of the vessels Nevertheless it should be added that conditions wholly apart from the state of the blood, and further, quite complex, contribute, each for its part, to favoring or impeding the production of vascular

murmurs, so that the appearance of murmurs is the result of multiple circumstances which cannot be revealed except by a careful analysis. Thus the inconstancy of the murmur in conditions apparently identical at least as to the state of the blood. Also the impossibility of finding a cause which is single and always the same for this phenomenon.

Gentlemen, permit me to begin this subject with certain developments. This seems necessary to me because directly opposite opinions are found here sustained by considerable authority. We must then seek and discover the reasons for these divergences which are surprising when it is borne in mind that it is only a question of establishing a plainly physical fact and of understanding what relations it may have to certain conditions of the blood.

I do not wish to repeat the history of this question here. Since the discovery by M. Bouillaud, who *first reported it*,* with wise restrictions, of the relationship habitually existing between intense murmurs and anemia, everyone knows that the most divergent views on this subject have arisen among physicians.

Some, such as Gorup-Besanez, Skoda, M. Chauveau, and recently in your presence, M. Peter, reject vascular murmurs absolutely from the symptomatology of anemic states; others, on the contrary maintain with MM. Barth and Roger that, "continuous *murmurs are the most certain* indication of advanced chlorosis and of anemia with diminution of blood corpuscles."

This question deserves examination because it arises constantly at the bedside and the solution may, in many cases, considerably influence our practice. But, in order to procure the necessary exactitude, it is necessary in anemia to distinguish two different states, the influence of which on the production of murmurs is certainly very different, thus: true anemia or the diminution of the volume of blood, and hydremia or the diminution of corpuscles.

A. *The Relation of Murmurs to True Anemia.*—In spite of the contrary statements of M. Vernois, M. Chauveau, and M. See, I do not believe that true anemia can give rise to the vascular sound which we are considering here, that is, continuous or intermittent venous murmurs. It is known that it is not at all possible to confirm the isolated existence of this form of anemia, except when a large hemorrhage takes place in a person with good health. Now I have practiced auscultation a large number of times in these conditions and I have never seen the murmurs of which we speak appear at that time when they had not been present previously. On the contrary, I have seen, as M. Bouillaud pointed out in his first work on vascular murmurs, the murmur disappear under the influence of a blood loss of some importance, to return much later, during the period of restoration. This morning on the obstetrical service of the Necker

*See page 446.—F. A. W. 1940.

Hospital, I saw a woman who had an abundant hemorrhage and who did not show any trace of a murmur over the jugulars Besides, do we not see every day that in those of our patients in whom this type of anemia predominates especially, the phthisical, the cachectic of any type, they are precisely those in whom the jugular murmurs show the least intensity? This first point appears proved to me, moreover, it is not in dispute Let us pass then to hydremia

B *The Relation of Venous Vascular Murmurs to Hydremia*—That which we now know of the relation of vascular murmurs to hydremia rests principally, nearly exclusively, on the researches of M Bouillaud and M Andral The analyses of Gorup-Besanez and certain ones which MM Becquerel and Rodier have made on the blood of chlorotics have been opposed to them already And now M Parrot and M Peter present their clinical observations to you as absolutely contradictory to the opinion of the masters whom I cite But there should be no contradiction in well observed facts, the contradiction can be only in the interpretation which is given them and in the deductions made Let us see then if we may discover them

M Bouillaud immersed the hydrometer of Baumé in the blood of twenty-seven persons who had been bled for various febrile affections, several successive bleedings In each individual, the murmur appeared and was increased according to the repetition of bleeding and the further depression of readings of the instrument Further, each time that the hydrometer showed more than 6¼ the vessels had no murmur, on the contrary, every time that the hydrometer fell below 5½, the murmur was present Between these two limits there was an equal number of subjects having the murmur and of others who did not show it But if these two groups of data are analysed further and one takes the reading of 5¾ for a median point, it is found that among those patients who showed a specific gravity of the blood above this figure, only one had a murmur and eight did not have it, while on the other hand, those in whom the density of the blood was less, seven had a murmur and only one did not From this one may rigorously conclude that, under the conditions in which M Bouillaud worked, that is to say in the patients who were bled, the murmur became better manifest as the blood was impoverished further by bleeding, that this sound appeared only exceptionally when the density of the blood taken with the hydrometer was above 5¾ and that it was almost constant when it descended below this figure By innumerable observations M Bouillaud has discovered that intense murmurs are almost always found in individuals, men or women, who show the signs of chloranemia to a high degree And as it is known, moreover, that the blood of chloranemia lacks especially red corpuscles, that it is the lowered proportion of the same corpuscles in the blood of persons subjected to bleeding that chiefly depresses the density

of that liquid, it results that chloranemics and patients bled in any large quantity are, in this respect, in the same condition, that they have blood equally poor in corpuscles and that this special poverty is accompanied equally in both by vascular murmurs, there should be in all cases a strict relationship between the presence of murmurs and this state of the blood.

But M. Bouillaud has not said in any manner and he has never contended that the murmur is an absolutely constant phenomenon in every degree and every condition of anemia, nor that it is never met with in persons exempt from this pathologic state, still less did he say that the intensity of the murmur should be strictly proportional to the degree of hydremia. Because, to use the master's own words, it is only when this phenomenon is present "in a high degree that one may affirm the existence of a chlorotic or anemic state, and reciprocally."

Likewise, the analyses of M. Andral have shown that in general the murmur is constant when the number of corpuscles falls below 80 p. 1000, that the murmur is likewise more frequent as the proportion of corpuscles, in falling, more nearly approaches this figure, that finally the continuous murmur is found more frequently in relation to the intermittent murmur as the more pronounced states of hydremia are observed. But this does not imply that murmurs always accompany anemia, that they do not exist without it, nor that they give an exact measure of it by their intensity.

There is nothing contradictory between these clinical rules and the observations which have been presented to you by M. Parrot and M. Peter. The observations of these gentlemen have shown us a certain number of persons without a murmur with the external characteristics of anemia or subjects who had intense murmurs with the appearance of excellent health. Now these facts, in some respects, being implicitly compromised by the rules in this regard which M. Bouillaud and M. Andral have established, since, according to them, the moderate degrees of anemia, those in which the figure is above 80 p. 1000, do not have the vascular murmur for a constant symptom. We accept them then, and we acquiesce so much the more willingly, for there is not one among us who has not, undoubtedly, been surprised more than once by facts of this nature. In collecting such a large number, our colleagues have given us a useful lesson for which we should be grateful to them. It will caution us against an error which we are, I believe, rather generally inclined to commit, that of easily granting, in practice, to this simple and easily determined sign, an absolute diagnostic value which it should not have, and of not taking enough trouble to determine its value by analysing the causes and conditions present. I have already made some efforts to combat this too common tendency in the article on anemia which I prepared for the *Dictionnaire encyclopédique des sciences médicales*.

Are we to say now that it is necessary to give up the view of relations established between the murmur and the hydremic state? Is it necessary to reject all of the positive results of experiments and analyses which I have just now cited? Is it necessary to seek the sole cause of abnormal sounds in circumstances independent of the state of the blood? For my part, I do not think so

Only one serious objection can be made to the analyses of M Bouillaud and M Andral, it is that they deal with a number of cases relatively too restricted for the exceptions to the law which they formulate to be able to show themselves, and there does remain some incertitude as to the value of murmurs in the diagnosis of anemias of moderate intensity, since they had for subjects, patients affected by various conditions which further complicates the question As to the contradictory analyses of Gorup-Besanez or of Becquerel, we may leave them aside because they deal with a number of cases even more restricted To decide the question finally, it is necessary then to make a very large number of analyses, especially relative to murmurs of medium intensity, bearing comparatively on persons in the physiologic state and on patients simply anemic For to estimate the state of the blood with some certitude merely according to rational symptoms, does not indeed require much thought We know that the external appearance of the patient is one of the least dependable of the signs of anemia M Bouillaud and M Andral have both sufficiently emphasized this fact But in order that the analyses may be practicable it is necessary to have a procedure for measuring corpuscles which can be used with absolutely insignificant quantities of blood But unfortunately we do not have such a one The procedure for enumeration proposed by Vierordt is not at all practicable, the more simple ones of Welcker, de Nasse and de Panum require too much blood, and the criteria which I have established to make these methods of research applicable clinically have not been met up to the present, with the inability to obtain instruments constructed with enough precision Montegazza in Italy, appears to have been more fortunate but he has not, to my knowledge, applied his instrument to this problem

I have then attempted to approach this problem in an indirect manner It is disputed whether or not the decrease of corpuscles in the blood is of any importance in the production of murmurs I wished to find out if blood flowing through an inert tube would produce more or less of a murmur according to the quantity of corpuscles present To that end I have constructed an apparatus which I have had brought before you and with which you may experiment in a moment It is made essentially, as you see, of a vertical tube of thin India-rubber of about the caliber of the internal jugular of an adult To this tube I have joined perpendicularly another of smaller caliber which, applied to it simply, does not communicate with its cavity This serves as a stethoscope, the other end be-

ing applied to the ear, and permits one to hear the murmurs which take place in the first tube without exercising the least pressure on it, without displacing it the least With a clamp of limited pressure, I can establish, at whatever point of the tube I wish, a narrowing of a perfectly determined degree and which I can vary at my will Finally, this tube is connected with a glass reservoir of known capacity which I can fix at any height above the point of auscultation To complete the apparatus two vessels, which may be put in communication with this common reservoir contain, one, serum of beef blood, the other, defibrinated blood, that is to say, serum with corpuscles The serum and defibrinated blood were taken yesterday at the slaughter-house from the same animal After having produced a certain narrowing with the clamp above the point of auscultation, at first I let pure serum run through the tube which immediately gives rise to a very intense murmur, then, while the liquid is still running, I substitute serum with corpuscles for the pure serum, and at the moment that the former comes into the tube taking the place of the latter, a considerable diminution of intensity of the murmur takes place Finally, each time that these two liquids are alternated, the murmur is heard stronger or weaker, according to whether serum or defibrinated blood is flowing One may, moreover, arrange things in such a way that the murmur produced by the serum is only of moderate intensity, then all sound disappears when the blood with corpuscles begins to flow If one uses not pure serum but serum mixed with a small quantity of corpuscles, as is the case in anemias, one still obtains the same results, but with proportional differences of intensity

Absolutely nothing changes here for the duration of the experiment except the proportion of corpuscles in the flowing liquid, and the murmur appears or disappears, increases or decreases constantly with each of these alternations Does this not demonstrate, in a most rigorous fashion that blood which contains the fewest corpuscles is most apt to produce vascular murmurs?

Experiments of the same type, with inert tubes, have been made by various workers, notably Weber, M Piorry and especially M Monneret But they have had the aim of explaining and not of demonstrating the aptitude of blood for producing murmurs, also the experimenters have used various liquids of different densities or viscosities, but not blood itself Here the demonstration is direct and should not, I think, allow any doubt on this particular point of the question that the quality of the blood, as to its greater or lesser content of corpuscles, *may* influence the production of murmurs, an influence absolutely independent of any vital act of contraction, dilatation, compression *etc*

But why does the flow of blood richer in corpuscles produce fewer murmurs? Is it because it is less apt to vibrate, as is generally believed and as is accepted by M Chauveau? Is it, as M Monneret believes, be-

cause it flows less rapidly? In this regard, the same apparatus will give us a solution and a precise demonstration. You have been able to note, when serum and defibrinated blood passed alternately, that the latter flowed more slowly. If I now change the height of the reservoir without changing any of the other conditions of the experiment, I can likewise change the speed of flow. Now, when we have arranged these changes in height in such a manner that the rate of flow is the same for the two bloods, that is to say that the quantity of liquid contained in the reservoir flows out in the same number of seconds, you shall see that the intensity of the murmur does not change and that it apparently remains the same, whatever the quality of liquid employed. In this instance, only one thing appears to me to differ, that is the quality of the sound, the quality being more subdued, lower, in the blood richer in corpuscles.

Thus, in this apparatus, the poor blood makes more sound because it flows more rapidly, and, when the flow of the richer blood is made equally rapid by increasing the pressure or suction which makes it move, the difference of intensity disappears, at least sensibly.

There is no reason to think that things happen otherwise in the blood vessels. On the contrary, there are reasons to believe that differences in velocity, due to the unequal proportion of corpuscles, are exaggerated considerably because of the multiplicity of obstacles which the capillaries present in place of the single obstacle which is in our apparatus. It must be admitted then that poor blood sounds more in the vessels, principally, and possibly solely because it flows more rapidly.

But you know, Gentlemen, that the velocity of blood flow in the living economy is not at all exclusively regulated by the quality of the flowing liquid. The force of cardiac impulsion, the width of the capillaries, the aid or resistance present in the chest are also conditions which strongly influence the course of blood in the vessels, and notably in the veins. We may add, that a local narrowing, followed by a relative dilatation, is necessary for the production of a murmur, as M. Chauveau has shown, the proof of which you may easily obtain in the apparatus before your eyes, that this narrowing, produced naturally or brought about by the instrument of auscultation, may vary by reason of a thousand circumstances, finally that a certain compression capable of slowing the flow of blood acting on the vessel either above or below the point of origin of the murmur, restrains or impedes its production. Thus you may judge, on the one hand, how different influences are added together and combined in order to unite to give rise to the phenomenon with which we are concerned, or to modify its manifestations, on the other hand, how the mode of action of these diverse influences probably amounts in all cases to causing variations in the speed of flow at the point of auscultation.

Thanks to these considerations, is it not now easy to understand how, within certain limits the presence of murmurs is connected with the existence of anemia, and how, nevertheless, this symptom cannot constitute a certain sign nor a measure of intensity

One of the proofs of this latter fact, which for my part has impressed me very much, is that which is found quite frequently in the course of typhoid fever Almost all persons attacked by this affection show a very marked murmur during the first period, that is to say the first septenary of their disease I might almost say all, because I can scarcely recall any exceptions to this rule, after having done auscultation on a very large number of patients It seemed difficult to believe that the murmur was here the expression of an anemia of any general type, or that typhoid fever would attack anemic persons almost exclusively But this difficulty is no more, a greater one soon arises In proportion as the disease progresses, in proportion as the turgescence of the face so remarkable in the first days disappears, in proportion as the patients become pale and lose their color, made anemic by the disease, by the diet and sometimes also by the method of treatment, also in proportion the murmur, far from being augmented as one should expect, on the contrary becomes weaker, is subdued, is diminished, and sometimes disappears completely Now, if the murmur of the first days was a pure symptom of anemia, there is no way of understanding how it becomes weakened and ceases while the anemia obviously increases We are obliged to admit that the sound heard at first did not arise from anemia, but rather from the state of the circulation, facile and rapid, in the first days, across the dilated and turgescent capillary system, on the contrary feeble and slow when the weakness of convalescence arrives

Now that we may attempt to picture to ourselves the combination of conditions which regulate the production of venous murmurs, I believe that they may be reduced to two principles (*a*) The rapidity of blood flow in the vessel where the murmur occurs, (*b*) The arrangement of the vessel itself and of the surrounding parts

a It is easy to find more than one convincing example of the increase in the venous murmur caused by acceleration of blood flow—For if, on auscultation of the jugular, one exerts a sudden pressure over a certain extent of this vessel above the point of auscultation, the murmur is completely arrested But, before disappearing it is reinforced, as a sort of a gush, because the pressure which forces out the blood contained in the vein makes it pass more rapidly beneath the stethoscope—Another example I have found in myself and in various patients that if, having placed the stethoscope over the inguinal region in such a manner as to hear the venous murmur (a murmur that can be heard wonderfully well in this region, although the contrary is generally maintained), if, I say, the muscles of the leg and thigh are made to contract forcibly at this moment,

there is heard, at the same instant, a very obvious continuous murmur, sometimes quite intense, which immediately fades away and disappears. This is because the muscles in their contraction, force out the blood contained in the veins of the inferior extremity and precipitate it toward the crural vein where the flow consequently undergoes a sudden acceleration. Now it cannot be a question here, as some have wished to believe, of a reflex caused by the phenomenon of effort. If this were so, the sound would appear equally on the two sides, whereas it never appears except at the root of the limb of which the muscles contract.—Finally if, after having let one of my arms hang for some time and become congested, the rest of the body being extended horizontally, I then raise it rapidly in the air, I feel a very pronounced thrill in the subclavian vein and I also hear an obvious murmur. Now the transitory thrill and murmur can result only from the acceleration of blood flow in the vein. Indeed, they cease after an instant and are not reproduced unless the experiment is repeated at intervals long enough for the blood to have time to accumulate again in the veins of the extremity in its lowered position.

As these special influences created by the experiment should be left aside when we come to auscultation of the neck practiced in view of diagnostic indications, we may assert that the rapidity of blood flow in the jugular veins depends especially on the three following conditions: (1) the force of cardiac contraction, (2) the greater or lesser facility of blood flow through the capillaries, (3) modifications of thoracic aspiration.

The first, the importance of which one cannot doubt, is quite difficult to appraise in practice, because we do not have any precise and positive method of measuring the energy of cardiac contractions. We can only cite examples, such as the disappearance of murmurs in the syncopal state. —The second is without question the most important of the three, that which exerts the greatest and most constant influence on the production and intensity of murmurs. It is that which is obviously at work when murmurs are intensified by the influence of high temperature, of certain fevers which impart a special activity to the peripheral circulation (typhoid fever, eruptive fevers, etc.) or even physiologic fever which follows muscular effort and works in the same way. We are concerned with it again where an intense cold weakens the venous murmurs and makes them disappear for the time being. Its mode of action is easy to understand, because, the greater the abundance of blood passing through the capillaries, the more it returns through the veins, so that the rapidity of venous blood flow is thus accurately regulated by the activity of the capillary circulation. Thus it is here, without doubt, that we must seek the cause of many of the changes which take place in murmurs, without apparent cause and without supposed modification of the composition of the blood, changes which have embarrassed M. Peter so much. It is here, I believe, that we must transfer the spasm imagined by Laennec, of which our colleague has taken the de-

fense. Here, however, spasmodic contractions of the vascular walls suppress the murmur rather than produce it as Laennec believed and as our colleague thinks with him.

With respect to this we must remember that the tonicity of the blood capillaries is not the only condition upon which the velocity of blood flow in these vessels depends. Further, we have shown experimentally, how the variable proportion of corpuscles may accelerate or retard the flow of blood in this part of the vascular system.

The influence of thoracic aspiration on the venous flow in the neighborhood of the thorax is a fact so well known and supported by so many proofs that it is useless to lay stress on it. As to the action on murmurs and its consequence one may easily establish in this, that inspiration frequently exaggerates them, perhaps gives rise to them, and that their increase is more marked as inspiration is made with more effort. The increase is such that quite frequently it is taken for a tracheal sound, and one can rid himself of this error in no better way than this, by pressing the vein above the stethoscope lightly, the vascular sound ceases completely, leaving only that which arises in the air passages. Expiration works in the opposite manner, and in proportion as it is done with greater effort. Also one never finds any murmur in asthmatic and emphysematous patients, in spite of the usually large volume of their jugular veins, one might say precisely on account of the distention which they show.

In short, every acceleration of blood in veins is favorable to the appearance of murmurs on auscultation, every slowing makes an obstacle, and the causes of acceleration or of slowing may be found: for one part in the energy of cardiac action, for another, in the activity of thoracic aspiration, in the third place, in the facility with which blood passes through the capillaries. Furthermore the latter depends on two things: first, the width and variable tonicity of these vessels, second, the degree of viscosity of the blood which results from the greater or lesser proportion of corpuscles present. But that is not all, and we shall see in the following section other additional influences arise.

b. Let us come now to influences which depend upon the state of the vein and of the surrounding parts. It is at first an incontestable fact, after the demonstration furnished by M. Chauveau, that the blood does not produce a sound in flowing through the vessels except when it encounters a narrowing, that is to say, a point over which it passes from a relatively narrow portion which it crosses rapidly to a much wider portion where it flows more slowly and under lower pressure. This condition is realized in the jugular vein at various points and in various ways, for example, at the inosculation of the vein with the brachiocephalic trunk, perhaps at the level of the insertion of the valves, or further, at points compressed by muscles or taut aponeuroses, or finally, and frequently, at the point of pressure of the stethoscope. The various positions as-

sumed by the neck certainly contribute to making the realization more or less complete It should be added that, in the position recognized the most favorable for the production of murmurs, a slightly exaggerated rotation compressing the jugular of the opposite side, consequently tends to increase the activity of the circulation in the one left free and on which auscultation is done It should be added that a very light contraction of the sterno-mastoid may, by compressing the vein, weaken or completely efface the murmur Also as everyone knows, a slight displacement of the head is sufficient to cause this sound to appear or to cease suddenly It is to this circumstance, frequently quite difficult to appreciate except with very close attention, that I believe it is necessary to attribute, in a large number of cases, the variability of abnormal sounds which has so much surprised observers, and which M Peter believes cannot be explained without admitting a spasm of the large veins —It goes without saying that the thickness of the soft parts which lie over the point of auscultation should also enter in some way into the intensity of the sound So that the anatomical disposition of the parts exerts, as we see, a very complex influence on the manifestations of the sign which we have studied

In the presence of so many different influences, near and remote, which unite with anemia in determining the appearance of vascular murmurs, or in modifying their intensity, one cannot expect to see this sign express faithfully the existence or the degree of alteration of the blood To constitute a sign of anemia it is absolutely necessary, as Bouillaud has said, that the sounds should be "very intense" It is necessary further, that one should be certain that no other circumstance independent of the state of the blood exist capable by itself of causing the appearance of the abnormal sound Without analysing the conditions which give rise to it, this sign is of no value, in this regard I am in perfect agreement with my colleagues

However, there exists a particular form of the murmur which I believe is quite characteristic of hydremia, it is the sibilant, musical murmur which M Bouillaud calls "the song of the arteries" What makes me think thus is that this form has never appeared at all except in persons having elsewhere quite positive signs of anemia, this is the form in which it appears after loss of blood

I have shown previously, in my inaugural thesis that when a subject has undergone any abundant hemorrhage, the murmur, absent at first, appears only after some time, it increases progressively during a certain number of days, then it decreases until it finally disappears It increases in proportion as the blood, at first restoring its serum, becomes relatively poorer and poorer in corpuscles It decreases when the corpuscles, in their turn reappear, returning the blood to its former composition Now, during these periods of successive increase and decrease, one sees the

murmur successively take on the intermittent form, then the continuous, then the musical to return then to unmusical continuity, to intermittence and it disappears The sibilant murmur, which is thus placed at the peak of this sort of curve, corresponds then precisely to the highest degree of alteration of the blood and consequently appears a little more significant than the other forms of abnormal vascular sound

CONCLUSIONS

Before ending this communication already long, wherein I have not pretended to solve all of the questions which the problem of vascular murmurs brings up, allow me, Gentlemen, to summarize in some propositions that which the facts and reasons which I have had the honor to present to you, appear to me to lay down the most essential and the most positive

1 Two kinds of murmurs are heard in the neck arterial murmurs and venous murmurs,

2 Arterial murmurs are intermittent, venous murmurs may be continuous, intermittent, or continuous with reinforcements,

3 The reinforcements or repetitions of the venous murmur have no other cause than intermittent accelerations of blood flow in the vein,

4 These accelerations and their relation with reinforcement of the murmur are, moreover, demonstrated in a positive manner by the depression which appears, at the moment they take place, at the point of the region which corresponds to the vein They result from successive aspirations set up in the venous system near the thorax by diastole of the auricle and ventricle, as the sphygmographic tracings show,

5 The murmur appears the more easily in veins in which the blood is poorer in corpuscles,

6 If the poor blood is more apt to produce murmurs, it appears to be nearly exclusively because it flows more easily and more rapidly

7 The appearance of murmurs in the jugular does not depend solely on the poverty of the blood, it is subordinate further to other conditions, some of which are local, others general All appear to act, directly or indirectly, so as to accelerate or retard the blood flow in the vein ausculted, or to produce the local narrowing necessary to cause vibration of the liquid,

8 One may not attribute any value to murmurs in the diagnosis of hydremia except when they show a rather greater intensity and when, moreover, one has taken into account circumstances independent of the state of the blood which might contribute to their production,

9 There is however a variety of vascular murmur the clinical value of which appears somewhat more positive and which seems to be connected more directly with hydremic alteration, it is the musical form of this murmur

1867

T. LAUDER BRUNTON

THE INTRODUCTION OF AMYL NITRITE IN THE TREATMENT OF ANGINA PECTORIS

SIR THOMAS LAUDER BRUNTON

(Courtesy St Bartholomew's Hospital Reports)

SIR THOMAS LAUDER BRUNTON

(1844-1916)

"He was a kindly Scot"

—*The British Medical Journal* (1916)

THOMAS LAUDER BRUNTON was born on March 4, 1844, at Bowden, Roxburghshire, Scotland He was the youngest son of James Brunton, a gentleman farmer. Brunton was educated privately and in 1862 began his medical studies at the University of Edinburgh He had a marked capacity for work and received many honors as an undergraduate In 1866 he received the degrees of Bachelor of Medicine and Master of Surgery, and in 1867 he was granted the degree of Bachelor of Science During the latter year he served as house physician under Prof Hughes Bennett at the Royal Infirmary in Edinburgh In 1868, after receiving the gold medal award for his thesis, "Digitalis with Some Observations on the Urine," he was granted his Doctorate in Medicine In 1870 he obtained the degree of Doctor of Science

From 1867 until 1869, besides doing some original research at Edinburgh, he spent part of the time in study at the leading medical centers of Austria, Germany and Holland under many able teachers including Ernst Wilhelm von Brucke, Isidor Rosenthal, Ludwig Traube, Willy Kuhne, and Carl Ludwig At Ludwig's new laboratory in Leipzig, Brunton derived the main inspiration of his scientific life There he worked on the independent contraction of the arterioles and capillaries, making some experiments on the effect of amyl nitrite and sodium nitrite

Brunton meanwhile experimented in his laboratory at Edinburgh on the influence of digitalis on blood pressure in animals His instrument for recording this pressure was a simple mercury column He also experimented on the effects of digitalis on his own blood pressure with the aid of the sphygmograph Brunton's facile use of this instrument led him to discover the elevation in blood pressure which accompanies angina pectoris By correlating the pathologic data thus acquired and by utilizing his knowledge of the pharmacologic action of amyl nitrite, he succeeded in discovering a remedy for angina pectoris In 1867 he published the results of his remarkable observations, and it is our privilege to reprint this classical contribution

Brunton went to London in 1870 and was elected to membership in the Royal College of Physicians He was appointed lecturer on materia medica and pharmacology at the Middlesex Hospital A year later (1871) the post of casualty physician at St Bartholomew's Hospital was vacated, and he was appointed to this office That same year his wish to be associated in a teaching capacity with a famous medical school was fulfilled by his being elected joint-lecturer on materia medica and therapeutics at St Bartholomew's Hospital Until 1875 the lectures were divided with Dr Frederick Farre and when Farre resigned, Brunton took undivided charge of them That same year he was appointed assistant physician at St Bartholomew's Hospital In 1874 he became editor of "The Practitioner"

Under Brunton's direction, the whole scope of the lectures changed He used physiology as the foundation for his teachings and based his results on experiments carried out on himself, on his pupils, and on animals When he lectured, the theater was always crowded with students and with teachers from other schools Brunton continued these lectures yearly as summer courses until 1901

In 1874, at the early age of thirty, Brunton was elected a fellow of the Royal Society in recognition of his admirable work on the physiology of digestion and secretion, on the chemical composition of the blood, and on the action of digitalis and

mercury He served as a member of the Council of the Royal Society from 1882 to 1884 and from 1905 to 1906, and was elected vice-president of the society the latter two years

Brunton, in 1878, was placed in charge of the newly established department for diseases of the throat at St Bartholomew's Hospital He held this position until 1880, when he was succeeded by Sir Henry T Butlin From 1880 until 1895 Brunton was associated with the out-patient department of the same institution In 1895 he became full-time physician at St Bartholomew's Hospital, and he held that post until 1904 He resigned in 1904 because of illness and was immediately appointed a governor of the hospital and made an honorary consulting physician

Brunton had a long and enviable professional career For several years he served as examiner for the Royal College of Physicians and was a censor from 1894 to 1895 In 1877 he gave the Goulstonian lecture on "Pharmacology and Therapeutics" In 1889 he delivered the Croonian lectures on "The Connection between Chemical Constitution and Physiological Action" He was appointed Harveian orator in 1894, and he chose for his subject "Some Features in the Physiology and Pharmacology of the Circulation"

Among other appointments he was a member of the committee assigned in 1886 by the local government board to examine Pasteur's treatment for hydrophobia In 1889 he was chosen by the editors of the "Lancet" to be a representative to repeat the experiments on the administration of chloroform that originally had been performed by members of a committee sponsored by His Highness, the Nizam of Hyderabad

Besides holding membership in the chief medical societies of Great Britain, Brunton was a member of many foreign organizations In the United States he was honored by the following societies the American Academy of Arts and Sciences, the Academy of Natural Science of Philadelphia, the College of Physicians of Philadelphia, and the American Therapeutic Society He was also a member of the Therapeutic Society of Paris and the Imperial Military Academy of Medicine of St Petersburg

He received the honor of knighthood in 1900 and was created a baronet in 1908 The Universities of Aberdeen and Edinburgh awarded him the honorary degree of Doctor of Laws, and the University of Dublin awarded him the honorary degree of Doctor of Science

Brunton made numerous contributions to medical literature, many of which were of a pharmaceutic nature From time to time he collected his writings and reprinted them in book form In 1886 he published a volume "On Disorders of Digestion, their Consequences and Treatment", in 1897 he published his "Lectures on the Actions of Medicine," and in 1901 appeared a collection of papers written between 1874 and 1901, called "Disorders of Assimilation"

In 1907 he published his "Collected Papers on Circulation and Respiration" Included in this group of his reprints is his classic on the use of amyl nitrite in the treatment of angina pectoris

His most important work was his "Textbook of Pharmacology, Therapeutics, and Materia Medica" This volume appeared in 1885, and by 1887 three editions of the work had been published

Brunton in 1879 married Louisa Jane Stopford, daughter of the venerable Edward A Stopford, archdeacon of Meath She died in 1909 His elder son, Major James Stopford Lauder Brunton, succeeded his father in the baronetcy on Sir Lauder's death in 1916 His second son, Dr Henry Pollock Brunton, a member of the medical profession, was killed in action in France on October 8, 1915

Sir Lauder died on September 16, 1916, and is buried in Highgate Cemetery in London

ON THE USE OF NITRITE OF AMYL IN ANGINA PECTORIS*

By

T. LAUDER BRUNTON B.Sc, M.B.

Senior President of the Royal Medical Society, and Resident Physician to the Clinical Wards of the Royal Infirmary, Edinburgh

FEW things are more distressing to a physician than to stand beside a suffering patient who is anxiously looking to him for that relief from pain which he feels himself utterly unable to afford His sympathy for the sufferer, and the regret he feels for the impotence of his art, engrave the picture indelibly on his mind, and serve as a constant and urgent stimulus in his search after the causes of the pain, and the means by which it may be alleviated

Perhaps there is no class of cases in which such occurrences as this take place so frequently as in some kinds of cardiac disease, in which angina pectoris forms at once the most prominent and the most painful and distressing symptom This painful affection is defined by Dr Walshe as a paroxysmal neurosis, in which the heart is essentially concerned, and the cases included in this definition may be divided into two classes

In the first and most typical there is severe pain in the precordial region, often shooting up the neck and down the arms, accompanied by dyspnoea and a most distressing sense of impending dissolution The occurrence and departure of the attack are both equally sudden, and its duration is only a few minutes

In the second class, which from its greater frequency is probably the more important, though the pain and dyspnoea may both be very great, the occurrence of the attack is sometimes gradual, and its departure generally so, its duration is from a few minutes to an hour and a half or more, and the sense of impending dissolution is less marked or altogether absent

Brandy, ether, chloroform, ammonia, and other stimulants have hitherto been chiefly relied upon for the relief of angina pectoris, but the alleviation which they produce is but slight, and the duration of the attack is but little affected by them

*Lancet 2 97-98, 1867

In now publishing a statement of the results which I have obtained in the treatment of angina pectoris by nitrite of amyl, I have to observe that the cases in which I employed this remarkable substance belonged rather to the second than the first of the classes above described

Nitrite of amyl was discovered by Balard, and further investigated by Guthrie,* who noticed its property of causing flushing of the face, throbbing of the carotids, and acceleration of the heart's action, and proposed it as a resuscitative in drowning, suffocation, and protracted fainting

Little attention, however, was paid to it for some years, till it was again taken up by Dr B W Richardson, who found that it caused paralysis of the nerves from the periphery inwards, diminished the contractility of muscles, and caused dilatation of the capillaries, as seen in the web of the frog's foot

Dr Arthur Gamgee, in an unpublished series of experiments both with the sphygmograph and haemadynometer, has found that it greatly lessens the arterial tension both in animals and man, and it was these experiments—some of which I was fortunate enough to witness—which led me to try it in angina pectoris

During the past winter there has been in the clinical wards one case in which the anginal pain was very severe, lasted from an hour to an hour and a half, and recurred every night, generally between two and four A M , besides several others in whom the affection, though present, was less frequent and less severe Digitalis, aconite, and lobelia inflata were given in the intervals, without producing any benefit, and brandy and other diffusible stimulants during the fit produced little or no relief When chloroform was given so as to produce partial stupefaction, it relieved the pain for the time, but whenever the senses again became clear, the pain was as bad as before Small bleedings of three or four ounces, whether by cupping or venesection, were, however, always beneficial, the pain being completely absent for one night after the operation, but generally returning on the second As I believed the relief produced by the bleeding to be due to the diminution it occasioned in the arterial tension, it occurred to me that a substance which possesses the power of lessening it in such an eminent degree as nitrite of amyl would probably produce the same effect, and might be repeated as often as necessary without detriment to the patient's health On application to my friend Dr Gamgee, he kindly furnished me with a supply of pure nitrite which he himself had made, and on proceeding to try it in the wards, with the sanction of the visiting physician, Dr J Hughes Bennett, my hopes were completely fulfilled On pouring from five to ten drops of the nitrite on a cloth and giving it to the patient to inhale, the physiological action took place in from thirty to sixty seconds, and simultaneously with the flush-

*Journal of the Chemical Society, 1859

ing of the face the pain completely disappeared, and generally did not return till its wonted time next night. Occasionally it began to return about five minutes after its first disappearance, but on giving a few drops more it again disappeared, and did not return. On a few occasions I have found that while the pain disappeared from every other part of the chest, it remained persistent at a spot about two inches to the inside of the right nipple, and the action of the remedy had to be kept up for several minutes before this completely subsided. In almost all other cases in which I have given it, as well as in those in which it has been tried by my friends, the pain has at once completely disappeared. In cases of aneurism, where the pain was constant, inhalation of the nitrite gave no relief, but where it was spasmodic or subject to occasional exacerbations it either completely removed or greatly relieved it. It may be as well to note that in those cases in which it failed, small bleedings were likewise useless.

From observations during the attack, and from an examination of numerous sphygmographic tracings taken while the patients were free from pain, while it was coming on, at its height, passing off under the influence of amyl, and again completely gone, I find that when the attack comes on gradually the pulse becomes smaller, and the arterial tension greater as the pain increases in severity. During the attack the breathing is quick, the pulse small and rapid, and the arterial tension high, owing, I believe, to contraction of the systemic capillaries. As the nitrite is inhaled the pulse becomes slower and fuller, the tension diminished, and the breathing less hurried. On those occasions when the pain returned after an interval of a few minutes, the pulse, though showing small tension, remained small in volume, and not till the volume as well as tension of the pulse became normal, did I feel sure that the pain would not return.

As patients who suffer from angina are apt to become plethoric, and greater relaxation of the vessels is then required before the tension is sufficiently lowered, I think it is advisable to take away a few ounces of blood every few weeks. When the remedy is used for a long time, the dose requires to be increased before the effect is produced. A less quantity is sufficient when it is used with a cone of blotting-paper, as recommended by Dr. Richardson, than when it is poured on a large cloth. From its power of paralysing both nerves and muscles, Dr. Richardson thinks it may prove useful in tetanus; and I believe that, by relaxing the spasm of the bronchial tubes, it might be very beneficial in spasmodic asthma. I have tried it in a case of epilepsy, but the duration of the fit seemed little affected by it. It produces relief in some kinds of headache, and in one of neuralgia of the scalp it relieved the severe shooting pain, though an aching feeling still remained.

While cholera was present in Edinburgh during last autumn, Dr Gamgee proposed it as a remedy during the stage of collapse, a condition in which there are good grounds for supposing that the small arteries, both systemic and pulmonic, are in a state of great contraction No well-marked case afterwards occurring in the town, he was deprived of an opportunity of putting it to the test, but it is a medicine well worthy of a trial, and should another epidemic unhappily occur it may prove our most valuable remedy

Edinburgh, July, 1867

1868

HEINRICH QUINCKE

DESCRIPTION OF THE CAPILLARY AND VENOUS PULSE

HEINRICH QUINCKE

(Courtesy Charles C Thomas)

HEINRICH IRENAEUS QUINCKE

(1842-1922)

HEINRICH IRENAEUS QUINCKE was born on August 26, 1842, in Frankfort-on-the-Oder, Germany He was the son of a well-known physician His father moved to Berlin and there young Quincke attended the gymnasium Quincke decided to prepare for a career in medicine after he had completed his academic training He studied at the Universities of Berlin, Wurzberg, and Heidelberg His teachers included the anatomist, Heinrich Muller, the Swiss histologist, Albert von Kolliker (under whom he published a paper on the ovaries in the mammal), the physiologist, Henry L F von Helmholz, the chemist, Robert W E von Bunsen, and the master pathologist, Rudolf Virchow

In 1863 he received his medical degree from the University of Berlin, and in 1865 he worked under Ernst Wilhelm von Brucke in physiology in Vienna Quincke then spent a period of observation in the hospital and in the clinics of Switzerland, Holland, France and England

He returned to Germany where he became Friedrich Theodor Frerichs' assistant in Berlin At the age of thirty he succeeded Bernard Naunyn as professor of medicine at the University of Berne, in Switzerland Five years later he accepted the professorship of medicine at the University of Kiel in Germany, where he continued to work until his retirement in 1908 He then moved to Frankfort-on-the-Main and during the First World War was still active as a practicing physician He died in May of 1922 at the age of seventy-nine Death came to him suddenly and quietly as he was seated at his writing table at his home in Frankfort

Quincke was interested throughout his long career chiefly in the problems of general pathology and physiology, and he made many important contributions to these fields of medicine, in addition to his contributions to neurology for which he is so well known

He was the first physician to study, in detail, the capillary and venous pulsations and to evaluate their significance in establishing the diagnosis of aortic insufficiency He made this study in 1866, and it is a pleasure to present to our readers in translation the classic account of his work which he published in 1868 In 1870 he published his observations on aneurysm of the hepatic artery In 1876 he contributed the section on the diseases of the arteries for the "Handbuch der speciellen Pathologie und Therapie" of Hugo Wilhelm von Ziemssen

Two outstanding contributions of clinical importance were published by Quincke which in their day did not attract notice The first of these appeared in 1875 in the "Berliner Klinische Wochenschrift," and was entitled "Über Vagusreisung beim Menschen" Therein he called attention to a phenomenon which today is of the utmost clinical value, namely, the observation that pressure on the carotid artery of the neck results in a slowing of the pulse and he explained that stimulation of the vagus nerve was the cause of this phenomenon

With Heinrich Hochhaus in 1894 in the "Deutsches Archiv fur klinische Medizin" he published a paper entitled "Über frustrane Herzkrankheiten" Therein Quincke coined the term "Frustraner Kontraktion" of the heart to explain the extrasystoles

in which the heart sounds are heard but no peripheral pulse can be felt In 1882 Eugen Dinkelacker published at Kiel a 27-page monograph containing an observation of scientific importance which he had noticed in the clinic of his chief, Heinrich Quincke, the monograph was called "Über acutes Oedem " Quincke later described this observation in more detail The condition is known as "Quincke's edema," and he was able to show its association with urticaria and erythema multiforme

Quincke's greatest contribution to medicine probably was his invention of a diagnostic procedure, the lumbar puncture [1] There can be no doubt that this technique contributed as much to medical advancement as did Auenbrugger's discovery of percussion and Laennec's development of auscultation Quincke not only emphasized the diagnostic value of the spinal puncture but also pointed out its therapeutic significance

Quincke made other observations in neurology which made him famous in this field In 1909-1910 he published his studies[2] on the pressure of the spinal fluid and discussed in detail diagnosis by examinations of the cells, chemical observations or reactions, and the immune biologic properties of the spinal fluid His interest in the physiology of the spinal fluid dated back to 1872, in which year he described the anatomic relationship between the arachnoid and subarachnoid space, the relationship of the optic nerve to the cranial cavity, the function of the choroid plexus, and studies on the ventricles of the brain

A most important contribution to neurology was Quincke's description of "meningitis serosa," a contribution first published in 1909 [3] Quincke made many other contributions to neurology, too numerous to mention

In the field of hematology, his studies concerning pernicious anemia, destruction of the erythrocytes, and the production of hemosiderosis were among the first published accounts and contributed much to modern understanding of the physiology of hemoglobin metabolism

Quincke, in his later years, described the connection between hemolysis of the erythrocytes and the formation of bilirubin On the basis of clinical and experimental data he made some important contributions to the problem of jaundice

It seems unbelievable that one man was able, in the span of his life, to make so many important contributions to medicine But Quincke made many more observations than those thus far mentioned He was probably the first to treat abscesses of the lung surgically, and in 1901 published a fundamental paper on surgery in the treatment of tuberculosis Furthermore, he contributed important papers on the treatment of diabetic coma, Addison's disease, amoebic dysentery, typhoid fever, peptic ulcer, and postural drainage in the presence of abscesses of the lung

These are but a few of the outstanding and original contributions which modern medicine owes to the great clinician and physiologist, Heinrich Quincke

[1] Dr Essex Wynter of England discovered lumbar puncture independently, about this time

[2] Published in Zeitschrift für Nervenheilkunde

[3] Quincke Heinrich Zur Pathologie der Meningen, Deutsche Ztschr f Nervenh 36 343-399 1909

II. OBSERVATIONS ON CAPILLARY AND VENOUS PULSE*

By

DR. H. QUINCKE

Assistant in Medicine at the University Clinic of Berlin

IT IS an acknowledged fact in physiology that the pulsations in the arterial system which arise in the heart, extend to the smallest arteries, and that the blood stream in the capillaries is not influenced in the same manner by the beat of the heart. This belief is chiefly based on microscopic observations of the capillary stream in the mesentery of various animals, in the web membrane of the frog and of the bat. Only when there is a diminished flow of blood in the veins, or when there is a marked reduction of the arterial pressure and slowing of the heart beat such as occurs in the dying animal, is a propulsive movement of the blood in the capillaries observed.

Only one such observation in man exists, that of Lebert (*Handbuch der practischen medizin* I, p. 725), who observed in a case of aneurysm of the aorta, systolic reddening and diastolic blanching of the cheeks, constituting an actual capillary pulse.

Cl. Bernard observed a transmission of the pulse through the capillary system to the vessels of the submaxillary gland, extending to the tributary veins, when, following section of the sympathetic, simultaneous stimulation of the lingual branch produced maximum dilatation of the arteries.

Pertaining to man, only a few observations on the venous pulse exist, those by King (*Guy's Hospital Reports* IV, XII) cited briefly by Stokes (*Diseases of the Heart*). The original, unfortunately, has not been available to me.

There are, however, places in the human body, where under completely normal conditions but more clearly under pathologic conditions, one frequently observes the transmission of the pulse wave from the heart reaching to the capillaries and then into the veins; these sites are the fingernails, hand, forearm and foot.

Insofar as the capillary pulse is concerned, it can be observed in one's own fingernails but still clearer in those of another in the region between the whiter anemic area and the redder more injected part of the capillary system of the nail bed, in the majority of persons examined

*Berl klin Wchnschr 5 357-359 1868 Translated by F A W

there occurs, synchronous with the beat of the heart, a to-and-fro movement of the margin between the red and the white portion, and one can be convinced that the exaggeration of the redness ensues a moment after the apex beat and is still definitely systolic and occurs rather rapidly, while the recessive movement of the reddened margin occurs more slowly, that is, a perceptible delay in the wave as seen by the eye, in the same manner that palpation and the sphygmograph reveal it in the pulse waves of the radial artery

But the fingernails of everyone do not show this aforementioned white zone, in plethoric persons with strong and frequent heart action and high arterial pressure, in warm outside air the nails are not uniformly reddened Under these circumstances a clearer zone in the nail can be produced by (naturally similar) pressure, or better, by elevating the arms, by the latter manipulation one has at the same time the advantage that the blood pressure in the vessels of the arms falls, and the increased pressure (with constant cardiac action) which the blood derives with every beat of the heart is greater than the average pressure existing when the hand is not elevated

Similar conditions, which are produced by elevating the hands, that is, increase of the anemic areas of the nails and lowering of the arterial pressure, commonly occur in anemic individuals therefore, in those the capillary pulse is usually more distinct and also visible without elevating the arm but, the activity of the heart must not be greatly diminished, and therefore it is most distinct in mild chlorotics and not in convalescents following severe diseases

Greater amplitude and great output of individual heart contractions favor the distinctness of the capillary pulse, while marked increase in the arterial tension and greater frequency of heart contractions are less favorable The capillary pulse will either be perceived more distinctly or (more frequently) less distinctly, according to the predominance of one or the other factor in fever and under excitement The presence of large and rapidly falling pulsations is demonstrated in an exquisite manner in insufficiency of the aortic valves, for that reason the capillary pulse is especially distinct Even in the horizontal position of the hand one observes a distinct and rapid to-and-fro movement of the margin between the red and white zones, and also with a uniform coloration of the nail and a lightning-like and momentary accentuation of the reddening, so that the mannner of the appearance and disappearance of the capillary pulse, is objectively as characteristic a sign of aortic insufficiency as the exquisitely abrupt pulse is to the palpating finger Surely it is not so constant, for here likewise, in the appearance of the capillary pulse under normal conditions, the necessary conditions are not always present, and the requisite softness and transparency of the nail and the appropriate degree of elasticity of the arterial system must be considered

It is, generally, impossible to say in which fingernail the phenomenon is most distinct, but it seems to occur most frequently in the index finger. The white zone is usually found in the third quarter of the nail, measuring from the matrix, and the pulsation is at times more distinct in the lower part, at other times in the upper part.

Up to the present time I have not been able to observe the capillary pulse in the toenail, the reason probably being the diminution of the pulse wave in the long arterial tube and the great hardness and thickness of the nails.

Noteworthy is the observation of Kolliker (*Gewebelehre*, pp. 121 *und* 580) that in the production of the capillary pulse in the nail bed the average diameter of the capillaries is 0.005-0.008, while elsewhere in the human body it is only 0.002-0.006.

I have been able only a few times recently to observe a propagation of the pulse wave through the capillaries into the veins in individuals without valvular insufficiency.

The first object of observation was my own hand. Here, I saw on several occasions, after the veins on the back of my hand were markedly dilated from great heat, a weak but unquestionable post systolic pulsation.

Much more distinct was the venous pulse in a 50-year-old woman, H., who entered the hospital owing to cholelithiasis. She had never complained of palpitation, the heart sounds were clear, there was perhaps slight hypertrophy of the left ventricle, but this could not be definitely determined, owing to the unusual rigidity of the thorax. The palpable arteries were somewhat rigid, the pulse full, quite resistant, the pulse waves not especially short but waning abruptly. An extraordinarily distinct pulse was visible in the distended and prominent veins of the very thin and redundant skin of the back of the hand and forearm, as well as in the cross anastomoses extending up to the middle of the forearms, which even appeared delayed as compared to the same radial pulse, and still more evident as compared to the carotid pulse. The capillary pulse in the nails, in this case, was furthermore very distinct.

A third case of venous pulse which first came under my observation during the last few surviving days of life, occurred in a robust young man, who while diving in water, struck his head on the bottom and suffered a paralysis of all the spinal nerves issuing below the fourth cervical vertebra (the necropsy revealed a fracture of the vertebra, with crushing of the cord). In the veins of the back of the hand extending to the middle of the forearm, as in the foregoing case, a post-systolic pulsation was distinctly observed. It is highly probable that here we were dealing with a paralysis of the vasomotor nerves, which together with the high temperature of the air produced dilatation of the vessels resulting

in the venous pulse Here is an analogy with the venous pulse produced in Bernard's experiment of the salivary glands

In addition to these three cases there were also individuals with insufficiency of the aortic valves in which the bounding and rapid pulse wave was transmitted into the veins, and indeed, I could observe this phenomenon in the four cases which I recently had the opportunity of examining, however, not in all with the same clearness, for here, as in the production of the capillary pulse, certain conditions are necessary the skin must be fairly thin and loose (especially not hydropic), the veins must be filled to a certain degree, but not under too great tension, and thence elevation of the arms or obstruction of the veins can cause the venous pulse in the hand to disappear

In several of the mentioned cases of venous pulse, the centripetal direction of the waves could be clearly demonstrated by compression of the venous branches in the mid-portion of the back of the hand as soon as an area was selected, and a well marked stagnation of the blood ahead of the compressed area was prevented by anastomoses, and it was not transmitted beyond Peripheral to the compressed area, the pulse endured, centrally it disappeared —Compression of the A brachialis (without obstruction of the venous flow) regularly abolished the venous pressure

Other than the hand and forearm, the venous pulse was observed only once, and that on the back of the feet This was the first case of venous pulse *a tergo,* observed in the medical clinic by Dr Riess He found in the case of a 15-year-old, poorly developed, markedly chlorotic girl, who probably suffered from heart disease but could not be diagnosed with certainty a loud systolic murmur over all the cardiac orifices, loudest over the pulmonary artery and the aorta, also moderate hypertrophy of both ventricles Perhaps a communication existed between the aorta and the pulmonary artery The arterial pulse was soft, somewhat accelerated, often with a thrill-like character when the arm was elevated —In the extraordinarily prominent venous network in the pale, parchment-like skin of both hands and the backs of the feet, a full, likewise, palpable pulse was observed, which completely disappeared with the gradual disappearance of the chlorosis and improvement of nutrition Recently I have had the opportunity of observing the capillary pulse in man in still another location other than the fingernails, namely, in the retina, and it was in the last of the previously mentioned cases of insufficiency of the aortic valves, still under my observation In addition to an extraordinarily strong arterial pulse in the retina, extending beyond the papilla, there was revealed by ophthalmoscopic examination when the patient was erect, a uniform systolic reddened and diastolic blanching of the optic papilla, which resulted from alternating complete and incomplete filling of the capillary network, the color change was

most distinct at the middle in the border of the physiologic cupping, whereas in the finger nails, the border between red and lighter red moved to and fro with the pulse The capillary pulse was not visible beyond the border of the papilla, even when present, because here the color change, owing to the dark background of the choroid, was not sharply differentiated Furthermore, neither eye revealed any other abnormality accounting for the phenomenon of pulsation

The diastolic pulse in the central vein of the retina, which is frequently normally present, was seen with great intensity, since the production of this pulsation has not been entirely explained, and is probably not the result of transmission through the capillaries, it will not be discussed further at this time

From the above observation, it is apparent that the pulse wave which originates in the left side of the heart, is not as extensive as generally believed and disappears in the small arteries

Under appropriate conditions, only partly pathologic, partly within the limits of normal, the wave is transmitted through the capillaries, even into the veins, and it is also probable that the capillary pulse can be observed in other places than those mentioned, for example, internal viscera rich in blood, such as the spleen and kidneys The hands are a good place for observing it, partly because of the superficial situation of the capillary and venous network and also because the peripheral areas have an extensive blood supply and relatively broad arterial tributaries, the marked change in volume when hot or cold, reveals how extraordinarily adequate the blood supply is

I wish to express my best thanks to *Geheimrath* Frerichs for his generosity in making available the material in his clinic for these observations

1870

SAMUEL WILKS

DESCRIPTION OF BACTERIAL ENDOCARDITIS

SIR SAMUEL WILKS

(Courtesy Annals of Medical History)

SIR SAMUEL WILKS

(1824-1911)

"Of all the boys that are so smart,
There's none so smart as Sammy,
He is the darling of our Art,
And Guy's his Alma Mammy"

—W L Braddon, quoted by W H White,
Guy's Hospital Reports, 1913

ON JUNE 2, 1824, Samuel Wilks was born at Camberwell, London, England, the second son of Joseph Barber Wilks, who was a cashier of the East India House

Young Wilks began his education in a dame's school Later he attended a boy's school at Camberwell Green At the age of eleven he was tutored by the Reverend Dr Spyers at Wallop When Dr Spyers was made head master at Aldenham, Wilks accordingly went to Aldenham, where he stayed for three years In 1839 he spent one year at University College School, and in 1840 was apprenticed to Mr Richard Prior, the family physician, of Newington

During the second year of his apprenticeship he attended the course on anatomy given under the direction of Mr Bransby Cooper at Guy's Hospital The next year he continued the study of medicine at Guy's Hospital When he completed his qualifications he passed the examinations for a diploma from the College of Surgeons and the Apothecaries Hall

Wilks decided to qualify for a medical degree and therefore matriculated at the University of London. When he completed his classical course, he studied medicine at this institution In 1848 he received the degree of Bachelor of Medicine and in 1850 the degree of Doctor of Medicine For his high scholastic average in obtaining the latter degree he received the gold medal

In 1851 Wilks became a member of the Royal College of Physicians, and in 1853 he was appointed physician to the Surrey Dispensary He was married in 1854 to Mrs Prior, the widow of Richard Prior under whom Wilks had served his apprenticeship

The appointment of Wilks as assistant physician to Guy's Hospital took place in 1856 At that time Richard Bright (1789-1858) was consulting physician and Thomas Addison (1793-1860) was one of the physicians on the staff That year, also, Wilks was elected a fellow of the Royal College of Physicians At Guy's Hospital, Wilks served as pathologist, making systematic post-mortem examinations He also lectured on pathology and was curator of the museum He became full physician in 1867

In 1859 Wilks published the first edition of his "Lectures on Pathological Anatomy" He did much to place the study of pathology on a scientific basis in England. Formerly, physicians often had performed necropsies in order to ascertain whether or not their diagnoses had been correct, Wilks encouraged physicians to conduct examinations routinely to learn more about disease

In 1863 a paper was published by Wilks in "Guy's Hospital Reports" entitled "On the Syphilitic Affections of Internal Organs" According to White, Wilks was

the first to notice that syphilis, in some cases, attacks the viscera Possibly as a result of that observation, he was elected a fellow of the Royal Society in 1870

Wilks was one of the first to report on what he called "arterial pyaemia," now known as "bacterial endocarditis" We are reproducing his classic description of that condition, originally published in 1870 in "Guy's Hospital Reports"

Wilks soon began to devote more of his time to study of the diseases of the nervous system His most original work in this field was published in the "Lancet" in 1872 It was a study of alcoholic paraplegia He was mistaken in attributing this disorder to changes in the spinal cord, but he was the first to associate abuse of the use of alcohol with a form of paraplegia Wilks published his comprehensive study, "Lectures on Diseases of the Nervous System," in 1878 A second edition of the book was published in 1883

In 1879, Wilks was appointed physician to the Duke and Duchess of Connaught That same year he delivered the Harveian Oration before the Royal College of Physicians

Wilks was an active member of the British Medical Association In 1872 he delivered the address in medicine at the annual meeting which that year was held in Birmingham He was president of the Section of Medicine at the meeting held in Cardiff in 1885, and in 1895, at the meeting held in London, he was president of the Section on Pathology and Bacteriology

In 1881, Wilks delivered an address before the Pathological Section of the International Medical Congress, which was held in London From 1881 to 1883 he was president of the Pathological Society

In 1884, Wilks received the honorary degree of Doctor of Laws from the University of Edinburgh In 1885 he was made a member of the Senate of the University of London That same year, at the age of sixty-one, he retired from the active staff of Guy's Hospital and was made a consultant

The Royal College of Physicians in 1896 elected him to the presidency of that organization A year later he was created a baronet on the occasion of the Diamond Jubilee celebration of Queen Victoria He also was made physician extraordinary to the Queen

Sir Samuel retired from active practice in 1901, at the age of seventy-seven He spent the remainder of his life in Hampstead, where he endured several severe illnesses He suffered from an inflamed appendix, which was removed He later was operated on for removal of an enlarged prostate gland He also suffered from a cerebral attack accompanied by unconsciousness which lasted two or three days This, in turn, was followed by paraplegia which confined him to bed for many months and prevented motion of the lower limbs In spite of his ill health, at the age of eighty-five he began to write his "Memoirs"[1] This work was finished and the proof was corrected by the time he reached the age of eighty-seven Wilks finally lay in bed completely paraplegic He died on November 8, 1911, and was cremated on November 11

[1]Wilks, Sir Samuel *A memoir by Sir Samuel Wilks on the new discoveries or new observations made during the time he was a teacher at Guy's Hospital,* London, 1911, Adlard and Son, 200 pp

CAPILLARY EMBOLISM OR ARTERIAL PYAEMIA*

By

SAMUEL WILKS

DID space allow I could report several cases of this disease exemplifying the constitutional symptoms attendant upon the passage of disintegrating fibrin through the system, but I will content myself with reminding the reader of the importance and frequency of the complaint, although it is one which is constantly overlooked It may be safely said that there has been no more important addition to pathological science than the doctrine of embolism, the facts included in it have been at once recognised by the profession as throwing a light upon cases which before were most obscure It is remarkable, however, that only one portion of the statements in Dr Kirkes' paper seems to have been generally apprehended by medical men,—that portion which alludes to the effects of the blocking of a large vessel by a vegetation carried from the heart such as the plugging of a cerebral vessel, and the attendant paralysis, or the plugging of an artery in a limb, and the consequent gangrene But there is another, and equally important, part of this paper in which he speaks of the blocking of the smaller vessels in the parenchymatous organs, with constitutional symptoms The results seen in the organs have long been known and described by Rokitansky under the term capillary phlebitis, but the severe and even fatal symptoms often attendant thereon have not yet been sufficiently recognised By the term embolism, I say, is generally implied the case of the blocking of a large vessel and the resulting local symptoms, but under it should be included the equally important and common case of the obstruction of the smaller arteries, with attendant constitutional symptoms I have for many years been in the habit of insisting upon this both in the post-mortem room and in the wards Formerly, I was accustomed to show from a strictly pathological point of view how changes occurred in the arterial system analogous to those which take place in the venous —that as in phlebitis some morbid matters, products of inflammation, being taken up by a vein and carried inwards through the circulation, give rise in the internal organs to depositions of a similar kind in them, so in the arterial system disintegrating fibrin of the blood may be carried from the centre of the circulation to the periphery, and there give rise to further deposits of a like fibrinous matter Latterly, I have been enabled to show clinically that in both

*Wilks, Samuel Select Clinical Cases, Guy's Hosp Rep (Series 3) 15 29-35 1870

cases there are attendant febrile symptoms with characteristic arthritic pains and occasional rigors, and that just as there is a venous pyaemia having its source on the outside of the body, so there is an arterial pyaemia having its origin within. The term "pyaemia" is, of course, not used in its strict etymological sense, but then it must be remembered, that a rigid application of the word is not required in the more ordinary case of contamination of the venous blood, it is for the objectors to use the term "septicaemia."

This form of affection, I believe, is far from uncommon, as pointed out in a lecture, of which an extract is given in *British Med. Journal* of March, 1868. That it is overlooked arises from the circumstance that a severe organic disease exists generally at the same time, and that this is considered sufficient to account for the symptoms and death. A patient, for example, is in hospital for valvular disease of the heart arising, perhaps, from rheumatism at some former period; whilst under observation he may have febrile attacks attended by articular pains, but these are regarded as touches of the primary complaint, and when after death the valves are seen covered with vegetations, and the spleen and kidneys full of softening fibrinous masses, these are in no wise regarded as having been instrumental to the fatal event, but as mere accidents of the disease. It must, however, have often, I believe, occurred to the observer that the derangement of the valvular apparatus was scarcely sufficient to cause death. I can myself recall more than one instance where a patient died several weeks after an endocarditis, and the cause was attributed to heart disease, but where the valves were apparently quite efficient. The cases above all others which afford the most striking examples of arterial pyaemia are those where the endocarditis has left the valves of the heart altogether structurally uninjured, and, therefore, where no mechanical causes resulting from heart derangement can possibly have produced death. Such an instance I mentioned in the lecture above referred to, where a medical man was seized with all the symptoms of pyaemia, in so marked a degree that the only question discussed by his attendants was the probable source of infection. This turned out not to be in the veins at all, but in the arterial system, where the origin of the disintegrating fibrin was found to be an aneurism in the auricle of the heart. Such a case was a simple one of death by arterial pyaemia without any organic lesion. Of course, this constitutional affection may be seen in conjunction with that of local embolism, as in a case I took to the Pathological Society, where a man, besides having in the profunda artery of the leg an embolus threatening gangrene, had articular pains in all the limbs, with febrile symptoms dependent on the circulation of smaller particles of deleterious matter through the system.

The occurrence of fibrinous masses in the solid organs of the body has long been known, and as early as the year 1832 a kidney thus affected

is portrayed in these Reports Rokitansky described the condition under the name of capillary phlebitis, and states his opinion that it is due to some spontaneous disease of the blood It was mainly to refute this that Dr Kirkes published his paper, in which he proved that particles of fibrin were carried to a distance from the heart, where they had previously been formed This constituted Kirkes' originality I have, however, never given up the opinion that in many cases the deposit may have occurred from a primary change in the blood itself, since often there is no proof of the existence of a primary endocarditis, in fact, there has been reason to believe that in some cases the deposit found on the valves of the heart has occurred simultaneously with the deposits in the solid viscera It must be admitted that if, on post-mortem examination, there be found associated with these deposits in the viscera some vegetations on the cardiac valves, it would be presumptuous to deny that an endocarditis might have been the origin of the whole train of subsequent events, yet, on the other hand, it would be a practical error not to be awake to the possible occurrence of arterial pyaemia, because there is no history of a primary cardiac affection For my own part, although Kirkes may be right in the main, I think there is every reason to believe that Rokitansky's statement is equally true, that deposits may occur from changes in the blood itself Practically I am sure the supposition of such an event will enable us to recognise cases otherwise obscure

The facts, then, are these—there is the simple case of endocarditis, or the case where vegetations are covering chronically diseased valves, and as a result the deposition of fibrinous material in the capillaries of organs and other parts of the body In such a case the symptoms and death by arterial pyaemia are, I believe, frequent enough, although not so generally recognised as they should be I would also insist that, irrespective of a history of a primary heart affection, such symptoms of pyaemia should lead us to a careful examination of the heart in all cases, when the existence of a bruit may at once suggest their true nature Whether in such a case the vegetations on the valves which are productive of the morbid sound existed previous to the formation of deposits elsewhere, and were the source of them, or whether they occurred simultaneously from a blood-change, is a question often as difficult to decide after death as before it Then, again, we may meet with cases where the patients present all the symptoms of pyaemia, and where, failing to find any source for the blood infection on the surface of the body to contaminate the venous blood, we may conjecture that the pathological processes are going on in the arterial system, even though we fail to detect any morbid sounds in the heart, for a post-mortem examination sometimes shows that the viscera are affected in the manner above stated, when the interior of the heart is altogether healthy

One reason I have for believing that in many instances the source of infection is not the carrying away of vegetations from the heart is, that in the obscurer kind of cases the effects are very slow in developing, whereas in the instances where large portions of fibrin are carried away in the stream of blood the effects are more sudden and momentous. In these chronic cases the disintegration of the fibrin is slower, the smaller vessels in the viscera are occluded, and the organs which suffer are other than those most usually selected in marked heart disease. Thus the spleen not only has isolated masses within it, but the whole organ becomes enlarged by the deposition throughout it. In the same way the liver may be enlarged and hardened, and also the lungs. If in such a case the disease of the blood were due, not to some morbid process originating in the fluid itself, but to a change wrought upon it by the lining membrane of the heart, I should conceive that the latter, although in a sufficiently unhealthy state to be competent to effect this change, was not covered with vegetations, so as to give rise to a bruit, or to account directly for the deposits in the organs by simple transmission. In time, of course, such vegetations might arise, and produce a murmur.

In these cases it is probable that the first symptoms which attract attention will be the constitutional ones, and that febrile symptoms and occasional rigors will suggest the existence of ague; in fact I have seen several cases of pyaemia, both venous and arterial, treated for miasmatic fever. After a short time it is possible that the liver and spleen may be felt enlarged, and still the obscurity remain. At a later period a bruit may be heard, which may be styled aortic or mitral, according to position. The febrile symptoms continue, and the patient, perhaps after a protracted illness, dies; the organs are found affected as described, and vegetations on the valves of the heart. There may be appearances suggesting an old cardiac disease, and accounting for the deposition of fibrin; but it is equally probable that there may be nothing in the heart to indicate an older change than that observable in the solid viscera. I have now seen so many instances of this, both in hospital and private practice, that I recognise them as belonging to a class, although I am often unable to state the origin of the blood change.

That endocarditis with vegetations on the valves is not necessarily a primary affection is seen in the fact that it may result from an ordinary venous pyaemia; for example, a healthy man may fracture his leg so as to necessitate its amputation; after a time he may have pyaemia, and then an endocarditis. In scarlatina, after the usual recovery, a secondary fever of the nature of a pyaemia is often seen, in which pains in the joints occur, and not unfrequently an endocarditis, so that it is not uncommon for an organic disease of the heart to be traced back to an attack of scarlatina. More than this, in post-mortem examinations of children

who have died of this disease, and even before the accession of well-marked pyaemic symptoms, fibrinous masses may be found in the spleen and kidneys

During the last few years, since my attention has been drawn to the subject, I have seen many instances of arterial pyaemia, and my mind reverts to cases occurring at an earlier period, and not at all explicable by the pathological doctrines of that day In a paper on Pyaemia in Vol 7 of the present series of these Reports, I relate a few cases where there was no evidence that endocarditis was the primary affection One was the case of a man who was admitted after discharge from prison, he was extremely low and depressed, and thought to be suffering from fever He shortly died, when the viscera were found full of masses of softening fibrin, and a vegetation existed on an aortic valve, but when this was removed the endocardium showed no evidence of inflammation Another case of Dr Habershon's, which had previously been reported by him in Vol 5, was that of a woman in a state of extreme poverty Being very ill she was sent to the hospital as a case of fever, the febrile symptoms were high, the tongue brown, the pulse quick, and there were daily rigors, followed by heat and sweating, no cardiac bruit could be heard She remained exceedingly ill, and was in that state which is usually called typhoid, when some blebs came out on the skin, and she had pains in all the joints and the rigors continued On the third day after admission a systolic murmur was heard, she gradually grew more prostrate, became delirious and died On post-mortem examination the spleen and kidneys were found full of softening masses of fibrin, and there was a slight roughness on the border of the mitral valve, as if vegetations might have once existed there In some cases, as I have said, the liver and spleen may be enlarged, and thus the observer's whole attention may be given to the abdomen I well remember how such a case, when I was a pupil, puzzled Dr Addison A woman for three months had had fever with rigors, supposed to be ague, and, with this, enlargement of the liver and spleen After death the heart was found diseased, although no evidence was given of it during life In looking through the "Transactions of the Pathological Society," I have no doubt that several cases of enlarged spleen were of the nature indicated, and as early as the year 1851, about the time when Dr Kirkes was engaged in his researches, a case was brought to the Society by Dr Hare of a young man, aet 25, who gave as his history that he took cold six weeks before, and that this was followed by pains in the joints, palpitation, and enlargement of the abdomen When under care he had a large liver, a large spleen, and albuminous urine, also oedema of the ankles, and a double murmur over the cardiac valves After death there were found vegetations on the aortic and mitral valves, the spleen and kidneys were much enlarged, hard, elastic, with a yellowish deposit

I would say, therefore, that arterial pyaemia is a by no means uncommon affection, and that it is seen frequently in chronic heart disease, but the symptoms are overshadowed by the more severe ones attendant on the valvular imperfection, or, if observed, regarded merely as rheumatic Also, that it may be often met with where there is no history of a primary heart affection, although an endocarditis at the time of the occurrence of the symptoms may exist Also that it should be suspected in cases of obscure febrile conditions, especially if accompanied by rigors, and more especially where the liver and spleen have been found to be slowly increasing in size

1872

LUDWIG TRAUBE

DESCRIPTION OF PULSUS ALTERNANS

LUDWIG TRAUBE

(Courtesy Charité Annalen)

LUDWIG TRAUBE

(1818 1876)

"Today, also, I maintain that experiment is the 'sine qua non' of scientific pathology Even therapeutics, I am convinced, will take a definite step forward when an attempt is made in a systematic way to modify the disease processes produced in animals by the well known drugs"

—Ludwig Traube in Introduction to his *Collected Works*

LUDWIG TRAUBE was born on January 12, 1818, in Ratibor in Silesia, Germany He received his academic training at the gymnasium in Ratibor, and at the age of seventeen he matriculated at the University of Breslau It was his father's wish that young Traube should become a physician, so he began the study of medicine at Breslau under the Bohemian physiologist, Johannes Purkinje (1787-1869), who at that time was the leader of medical thought in Breslau

Two years later, Traube went to Berlin, where he was attracted by the magnetic personality of Johannes Muller (1801-1858) It is said that the general state of medical education in Germany at that time was low and Traube, being greatly disappointed, decided to give up his medical career His father, however, insisted that his son continue his medical studies, which the young man did He devoted as much time as possible to the study of French medicine, which during this period was outstanding because of the teachings of François Magendie (1783-1855) and René Laennec (1781-1826)

In 1840 Johann Lukas Schonlein (1793-1864) came to Berlin He greatly impressed Traube, who later (1849) was to become Schonlein's assistant Meanwhile, Traube decided to study at Vienna where Karl von Rokitansky (1804-1878), the famous pathologist, and Josef Skoda (1805-1881), who introduced Laennec's method of auscultation into the medical curriculum, were his teachers Traube received the degree of Doctor of Medicine in 1841 but remained in Vienna for some time as a graduate student

Traube later returned to Berlin to practice medicine He had decided to devote himself to scientific research, but met with much opposition from various persons At that time the Charité was the only public hospital in Berlin It was exclusively controlled by the army, and in order to use the hospital Traube was obliged to become an assistant to an army physician He practiced medicine in a suburb of Berlin, and being accustomed to study cases that demanded scientific investigation, he frequently was obliged to pay for permission to perform a necropsy

Even though they were in many ways deprived of clinical resources, Traube and other young physicians met regularly once a week to discuss and read the outstanding researches of Magendie and Claude Bernard (1813-1878)

Soon Traube's ability and skill in the newer diagnostic methods of auscultation and percussion were manifested and many young medical students requested him to lecture to them in private courses Although his pupils were enthusiastic about their teacher, patients were annoyed by being subjected to too many examinations

and a regulation was passed by the Charité, directed against Traube, which stated that army physicians were required to do their own work without the help of assistants.

Being unable to continue his clinical studies at the bedside of patients at the Charité, Traube, greatly influenced by the famous experimental studies of the French physiologists, decided to turn to the exact methods of animal experimentation. After two years of study he published his first paper, "On the Causes and Origin of Those Changes Which the Lung Parenchyma Suffers After Section of the Vagi." This classic work brought him into contact with Rudolf Virchow (1821-1902), who was then assistant pathologist at the Charité. A lifelong friendship began.

In 1846 the first number of a journal originated by three friends (Traube, Benno Reinhardt, and Virchow) was published. It was called "Beitrage fur experimentelle Pathologie und Physiologie," and was superseded in 1847 by "Virchow's Archiv." This event marked a revolutionary milestone in German medicine. In his introduction to the first issue, Traube demanded a different approach to the study of scientific medicine, an approach based on experimentation and its correlation with clinical experience. In the second number of this journal (1847), appeared the famous paper by Virchow on the occlusion of the pulmonary artery and the consequences thereof. Traube also contributed his classical account on suffocation to that issue of the publication.

Traube's primary aim—to contribute to the advancement of modern clinical medicine—was not fulfilled, however, until 1849, when Virchow made it possible for him to receive a hospital appointment. At that time he became an assistant to Schonlein. In 1857 he became an associate in the Charité and was appointed an assistant professor at the University of Berlin, where his reputation as an outstanding physician soon became known. At the University of Berlin he became the leading teacher, but not until 1872 did he receive his full professorship.

During the years previous to his association with the University of Berlin, Traube experienced a period of great disappointment and several times was tempted to ask for his release. It was a battle against the political factions of the state directed against Traube mainly because he was a Jew. Only the personal friendship of sincere colleagues gave him enough moral support to withstand repeated attacks.

Traube's scientific contributions are contained in three volumes.[1] The first includes his physiologic studies and the second and third his clinical and pathologic researches.

Among the different papers in the first volume his studies on asphyxiation and his pharmacologic researches on the action of digitalis, curare, nicotine, bile salts, and potassium nitrite, must be mentioned.

The variety of outstanding contributions Traube made in clinical and pathologic research as reflected by the last two volumes of his collected works is remarkable. His studies on diagnostic procedures in relationship to diseases of the chest (Traube's semilunar space and Traube's double sound) have become associated with his name. These volumes, too, contain his important studies on bronchitis, abscess, and gangrene of the lung and the invasion of the lungs by particles of coal.

In the field of cardiology, two of Traube's papers are outstanding. In 1856 he published a comprehensive account, "Über den Zusammenhang von Herz und Nieren Krankheiten," in which he discussed renal lesions caused by passive congestion of cardiac origin. He separated this condition from the inflammatory group of kidney lesions observed in the presence of Bright's disease. In the second part of this paper Traube described the symptomatology of contracted kidney and explained

[1]Traube, Ludwig. *Gesammelte Beiträge zur Pathologie und Physiologie,* Berlin 1871-1878, A. Hirschwald, 3 v. in 4.

the associated cardiac hypertrophy as being the result of increased circulation observed by the diminution of renal capillaries

In 1871 Traube read his classic paper on ''A Case of Pulsus Bigeminus'' before the Berlin Medical Society It is our privilege to present this report to the reader in translation The report contains the first clear picture of pulsus alternans

Many honors came to Traube late in life as acknowledgment of his great scientific contributions in the field of medicine But whatever the honor paid him, equally great in his regard was the admiration of his pupils and associates who saw in him one of the foremost physicians of the time

At the age of fifty-six, Traube suffered, as so many physicians before and after him have suffered, repeated attacks of angina pectoris Finally congestive heart failure developed, to which disease he succumbed in April of 1876 at the age of fifty-eight

Traube, a true scientist, diagnosed his own disease as follows ''Hypertrophy and dilatation of both ventricles with arteriosclerosis and coronary sclerosis with partial fatty degeneration of the myocardia ''

A CASE OF PULSUS BIGEMINUS, INCLUDING REMARKS ON THE ENLARGEMENT OF THE LIVER IN VALVULAR INSUFFICIENCY AND ON ACUTE ATROPHY OF THE LIVER

By

PROF DR L TRAUBE

IN MY experiments on animals I have for years been acquainted with a type of pulse, which I have named "pulsus bigeminus" The observations relative to this are unfortunately somewhat scattered, but are available in my collected "Contributions to Pathology and Physiology"

The nature of the pulsus bigeminus may be said to be this following every two pulses which originate in the aorta, a longer pause ensues This phenomenon is differentiated from the pulsus dicroticus by the fact that *in the latter there is only one contraction of the heart* for every two beats of the pulse, while in pulsus bigeminus there are two contractions of the heart, which follow one another rapidly and are separated *from the preceding and succeeding contractions by a longer pause* For every two beats of the pulsus dicroticus there occur, as in the normal pulse, only two heart tones, while in pulsus bigeminus four heart tones are audible An accurate reproduction of the pulsus bigeminus is found in Table 9 under C in the first volume of my "Contributions" This was an instance of an animal that had been curarized and then poisoned with potassium cyanide, and shortly after being poisoned, the vagi in the neck were sectioned We observed the pulsus bigeminus appearing soon following section of the second vagus, after the pressure and pulse rate were increased in consequence of the operation

The first time that I observed this phenomenon was when I permitted the longer suspension of artificial respiration in curarized animals, when beginning failure of the left ventricle occurred One soon sees a marked excitation of the inhibitory spinal nervous system after the onset of suspension under the rising tension in the aorta, which is evidenced by a considerable diminution of the frequency of the pulse and an increase

**Ein Fall von Pulsus bigeminus nebst Bemerkungen uber die Leberschwellungen bei Klappenfehlern und uber acute Leberatrophie* Presented at the Berlin Medical Society on March 20, 1871 Published in Berl klin Wchnschr 9 185-188, 221-224, 1872 Translated by F A W

in the pulse volume, later, under reduction of the arterial pressure, excitation of the inhibitory spinal nervous system produces paralysis in the same place, the pulse frequency accelerates, and then under further reduction of arterial tension the pulsus bigeminus appears, it first follows each pulsus tardi which manifests overwhelming fatigue of the left ventricle preceding its complete standstill.

Through greatly prolonged suspension of artificial respiration, which was undertaken in the animals with sectioned vagi, and where the heart had been deprived of the influence of the inhibitory spinal nervous system, we see the pulsus bigeminus appear under circumstances similar to those in which the vagi are intact, that is, after the arterial tension is appreciably reduced and shortly before considerable reduction in the frequency of the pulse takes place, the low pulsus tardi appears, which portends the failure of the left ventricle.

Finally, I discovered that this phenomenon would appear every time, shortly after poisoning in an animal with the vagi sectioned, when a substance was administered which stimulated the heart through the entire inhibitory spinal nervous system.

I concluded from these facts, that two conditions are necessary for the appearance of the pulsus bigeminus.

(1) The heart must be released from the influence of the inhibitory spinal nervous system, and also

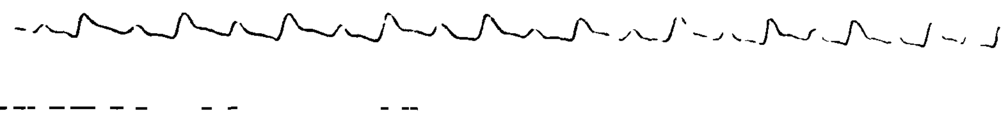
(2) There must be some agent circulating in the blood, which increases the irritability of the cardiac component of the inhibitory spinal nervous system, which is still functional.

If this conclusion is correct, we can obviously conclude from the appearance of the pulsus bigeminus in patients that a paralysis of the inhibitory spinal nervous system exists, and the prognosis under such circumstances would be unfavorable.

Up to the present time, I have been enabled in three or four cases to demonstrate the pulsus bigeminus in man, in two of these death ensued shortly after its appearance.

The following case, which came under my observation toward the close of last year, demonstrates a variation of the pulsus bigeminus, I designate it with the name of "pulsus alternans." It has certain features in common with the pulsus bigeminus in that the normal rhythm is not replaced by an arrhythmia but by a new extraordinary rhythm in which two consecutive pulses are in closer approximation to one another. It involves a succession of high and low pulses, in such a manner that a low pulse regularly follows a high pulse and this low pulse is separated from the ensuing high pulse by a shorter pause than that between it and the preceding high pulse. The following curve taken by me with the aid of Marey's sphygmograph from the radial artery of a patient, which forms

the starting point of this communication, gives us a more precise idea of this type of pulse



I present the case history in detail, since it appears more interesting in its various aspects

Observation

H W , laborer, forty-seven years old, entered my service on October 16, 1871, had for years engaged in heavy labor and was acknowledged an intemperate person He had smallpox a long time ago and recovered from pneumonia seven years ago At Christmas time 1870, he held a heavy barrel of spirits against his chest, which, together with other laborers, he attempted to carry up a stairway He felt no distress at the time, but in a short while he became short of breath, which so rapidly increased in intensity that he was obliged to discontinue the work he was engaged in, but also consumed fully two hours to cover the short distance to his home After the patient had remained in bed for eight days, he attempted to resume his occupation, which precipitated an exacerbation of the dyspnea which soon again returned him to the sick-bed, however, he suffered from neither cough nor pains in the chest After a month he felt considerably improved so that he again believed himself able to resume work, which he now pretended could be carried out without discomfort This condition of subjective well-being lasted until June, 1871 At this time, in consequence to more strenuous effort, great air-hunger occurred, but again without cough or pain in the chest Renewed forbearance of activities brought no relief this time Soon after this he began to complain of a disagreeable feeling in the epigastric region He was thereby moved to seek aid in Charity Hospital

On admission on October 16 the following was noted

Patient complains of air-hunger and pains in the epigastrium, in addition, marked objective dyspnea is present, cough is absent

Temperature, 37 8
Pulse frequency, 120
Respiratory frequency, 40

Orders One spoonful of castor oil, meanwhile a mustard plaster, and Sol Potas acetate, 10 0 in 200 0, 1 teaspoonful every two hours

A more thorough examination on June 18 revealed the following

The patient has a fairly robust constitution, a fairly generous panniculus, good musculature The dyspnea has become definitely decreased The thorax is well developed, although the excursions are limited There

is no cough Below the scapulae are occasional rales The cardiac dulness is increased in all directions and abnormally intensified The apex beat is distinctly visible and palpable in the sixth intercostal space, is one inch in breadth and lies beyond the mammary line Auscultation in the region of the apex beat reveals a loud systolic murmur, and a low diastolic sound, in addition to an otherwise normal tone, which accentuates the diastolic pulmonic arterial tone The radial artery was narrowed, from abnormal tension, low pulse The jugular veins distended, the liver greatly enlarged Urine scant, specific gravity 1 015

The temperature also remained normal during the ensuing days, while the frequency of the pulse varied between 116 and 124, the urine continued to be scant and red

On October 19, the infusion of digitalis was ordered, 1 0 to 200 0, 1 teaspoonful every two hours

On the twenty-second, after the use of 2 Gms of digitalis, the frequency of the pulse dropped to 104, the respiration to 24, the 24 hour urinary output rose to 2,000 c c, the specific gravity remained the same, 1 014, the temperature 37 1

Order Digitalis discontinued, instead, the patient is to be given a solution of potassium acetate 3 0 to 150 0, one teaspoonful every 2 hours

I personally first carefully examined the circulatory apparatus on the thirtieth of October This examination disclosed the following

The apex beat in the sixth intercostal space, near but beyond the mammary line, the area 1 inch in breadth, a second, but weaker systolic elevation is found, situated more to the inner side, in the fifth intercostal space Beyond the apex beat, observed at a distance from its abnormal location, there also appears an abnormal pulsation, consisting of a diffuse systolic elevation of the entire precordial region, which also involves the lower portion of the sternum and is most marked in the region of the costo-cartilaginous articulations One observes a systolic retraction at the point of the epigastrium, and another in the fifth and sixth intercostal spaces near the previously described elevation, in the neighborhood of the apex of the heart Auscultation in the region of the apex-thrust reveals a moderately loud systolic murmur, and a fairly loud diastolic tone, over the xiphoid process, two loud tones, and in the vicinity of the pulmonic area, a systolic murmur and a definitely accentuated diastolic tone Radial arteries quite broad, abnormally tense When one palpates the artery more firmly, distinct regular alternations in the height and depth of the pulse are noted

Pulse frequency, 96
Respiration, 20
Temperature, 37 3

Urine volume 2,000 c c, specific gravity 1 014

I made the diagnosis of hypertrophy and dilatation of both ventricles associated with sclerosis of the aorta. The pulsus alternans was the same as on preceding days.

Under the influence of an increasing icterus, which finally reached a profound degree, an increasing accumulation of abdominal fluid, persistent restlessness and delirium, marked collapse finally ensued. Death occurred on December twenty-seventh.

The post-mortem examination conducted twenty-four hours later (Dr. Wegener) revealed the following:

There was marked edema of the lower extremities, considerable ascites. The skin was of an olive color, and the visible mucous membranes intensely icteric. There was an area of hemorrhagic infiltration in the conjunctiva of the right eye. The ascitic fluid was of a yellowish-brown color.

The diaphragm on the right side reached the lower edge of the fifth, and on the left the upper edge of the seventh rib; in the left thoracic cavity was a copious serous, icteric-colored exudate, also considerable fluid in the pericardium.

The heart was greatly enlarged in all dimensions, its musculature quite firm; it contained a great quantity of fluid blood intermingled with clots. On the anterior wall of both ventricles were large fibrous patches; both ventricles were greatly dilated and hypertrophied. On the inner surface of the left ventricle, closely adherent to the trabeculations, was a thick continuously adherent, laminated, cystic and softened thrombus, of an icteric color, which in part was a dirty brown and in its inner portion of a greenish-yellow color. The valves, with the exception of having a pronounced icteric color, were normal. The ascending limb of the aorta immediately above the valves was dilated; in the upper portion of the arch, a prominent parietal thrombus was firmly adherent to the intima.

Epicritical Remarks

I. In this detailed case history, it is possible without difficulty to distinguish five periods of time.

In the first period the illness had its inception, that is to say, its latent course, at which time the hypertrophy of the left ventricle was so well compensated that the patient experienced no appreciable discomfort and his condition could have been recognized only fortuitously by even a meticulous and expert observer.

The second period begins with the catastrophe, the patient, together with other laborers, carried a heavy burden held against his chest, up a stairway This period is characterized by the fact that the patient became annoyed by air-hunger, at first only under strenuous exertion, later at rest, which was accompanied by a disagreeable discomfort in the epigastrium As the patient, on this account, sought admission to the Charity Hospital, one interpreted the signs, namely, a pulse of high frequency, that ranged between 116 and 120, marked narrowing of the radial arteries, weak pulse, distention of the jugular veins, marked enlargement of the liver and scanty red urine of high specific gravity, but without a trace of hydrops, as occurring with the appearance of dilatation and hypertrophy of both ventricles One was obliged to conclude, that it resulted from a loss of the compensatory mechanism of the left ventricle, leading to an engorgement of the pulmonary circulation, that as a result of this engorgement, hypertrophy of the right ventricle occurred, and finally, as the increased activity of the right ventricle began to fail, an engorgement of the systemic veins occurred

During the third period, which had its inception shortly after the onset of the illness, and when I could express myself, the patient's condition was generally improved by digitalis He walked about the room, participated in the care of other patients, complained only of short periods of palpitation, the presence of disturbance of compensation was evident only by the enlargement of the liver and the distention of the jugular veins In this period, which reached from October twenty-second to November seventeenth, we came upon a characteristic pulsus alternans

The fourth period began when the urinary volume again became diminished Then predominantly digestive disturbances occurred the patient complained of nausea, anorexia and diarrhea Later, the respiratory apparatus became involved attacks of air-hunger appeared, persistent cough with slimy, reddish colored expectoration, that ultimately became a dark reddish-brown, revealed the presence of hemorrhagic infarcts, now for the first time edema of the lower extremities appeared At this stage, the definite, unmistakable pulsus alternans that earlier was present in the carotids disappeared

The fifth stage extended from the thirteenth to the twenty-seventh of December (the latter being the day of death), and was primarily characterized by intense irritation of the entire digestive tract the diarrhea became more marked, added to this was violent recurrent vomiting, also icterus, and then the enlarged liver began to diminish in size During the rapid diminution in the volume of the liver the patient developed an agitated delirium, which simulated an acute mania, during which time the icterus increased in intensity, and extravasations appeared in the conjunctiva and in the skin of the abdomen Death occurred during the delirium and following severe collapse

According to the accustomed view, all the complaints could be attributed to arteriosclerosis, as revealed by the post-mortem. In this observation, it cannot be denied, that at least in a series of cases, the sclerosis occasioned conditions which hampered considerably the emptying of the left ventricle. It will thus be the case when as the result of degenerative dilatation of the aorta the elasticity of the walls of its greater arterial tributaries becomes diminished. Furthermore, these vessels assume the role of rigid tubes, and the force must be conceivably greater than ordinary, in order to deliver a specified volume of blood to a given place. But in the great majority of cases such a change in the large arteries does not occur, notwithstanding considerable hypertrophy and dilatation of the left ventricle and without other significant findings in the body which could be attributed to the disease of the heart, we find only slight or moderate degrees of sclerotic prominences in the lining of the aorta, which could not appreciably affect the elasticity of this vessel. Finally, one encounters cases, which in their clinical course, completely coincide with these, and in which obviously identical etiologic factors were at work, the post-mortem, with the exception of dilatation and hypertrophy of the left ventricle, revealed no changes in the large arteries.

One observes from this that arteriosclerosis could not possibly be the direct cause of the affection of the left ventricle, as is generally believed. It was Senhouse-Kirkes,* who first called my attention to the premise, that arteriosclerosis is the result of protracted high-grade tension in the aorta. Therefore, I believe, the first clue to a correct insight of the affection, is not only an understanding of the manner of its production, but also of its pathologic significance. If I did not completely accept the opinion of the authors mentioned in the foregoing discussion, and thus doubt them, that the abnormal tension in the aorta directly favors the development of arteriosclerosis, then I may also assert, that arterial disease as a rule is not the cause of the heart affection, but rather more often that both conditions are common co-effects.

Arteriosclerosis and hypertrophy of the left ventricle, especially in the aforementioned older persons, occurs particularly, according to more recent English experience, when the abuse of alcohol is combined with muscular exertion. If we examined such individuals more closely, the signs of abnormal tension in the aorta are soon evident. And the latter are apparent in those individuals in whom no degeneration of the aorta or its tributaries is disclosed by post-mortem. The basis for hypertrophy of the heart under these circumstances is readily seen, it can be none other than the increased arterial tension. Since the left ventricle empties its content into an arterial system of such great tension, that is, since the ventricle must raise the blood mass to a greater height than under normal

*See page 472.—F. A. W. 1940.

conditions, its bulk must increase, like all muscles, so that it is forced to perform greater work in order to effect normal conditions of nourishment According to Senhouse-Kirkes, as we have seen, arteriosclerosis here has the same cause as hypertrophy of the left ventricle, but I have recorded on an earlier occasion (No 29 in the *Wochenschrift* of the previous year), that in cases with which I dealt, yet another condition must be considered than abnormal tension of the arterial distribution, this is the slowing of the blood stream Inasmuch as alcohol probably increases the tone of the arterial musculature, and thus lessens the outflow from the aorta, it not only increases the tension in the arteries but also diminishes the rapidity of the arterial blood stream I have expressed myself in such detail in the last consideration on how the origin of the sclerosis may be favored, that I abstain from further explanations here regarding this belief, which in all events is evident, also, according to my concept, hypertrophy of the heart and arteriosclerosis are common basic conditions from which on the one hand the increased tension in the aorta results and consequently, hypertrophy of the left ventricle, while on the other hand, slowing of the blood stream in the larger arteries occurs, and consequently, upon this the sclerosis is dependent

Cases of this type, regardless of pulsus alternans, are more frequently observed in the hospitals but also are not infrequently encountered in private practice among the middle classes Here, as a rule, excessive muscular exertion plays no noteworthy role, and likewise, the abuse of alcohol is not of constant etiologic importance According to my experience, two other influences prevail excessive smoking of tobacco and congestion of the portal system, which become aggravated by sedentary living and over-eating I believe it is possible to approach these points by other means

Strange and therefore unbelievable as it appears in our case, the illness first began as the patient rolled the heavy keg of spirits against his chest As the post-mortem showed, there were no traces of partially healed severe acute disease of the respiratory or circulatory apparatus in the body, and herein, the complaints of the patient harmonize, since he complained only of dyspnea which appeared soon after the catastrophe, but without cough or pain in the chest From the standpoint of fever, as we see, there was likewise nothing to mention It required but slight reflection to understand, that the subtle events which we noticed did not indicate signs of disease, but indicated disturbance in compensation, in other words at the time of the catastrophe, while the patient was under the influence of excessive and frequent use of alcohol and repeated and unaccustomed muscular exertion, hypertrophy of the left ventricle, as well as the arteriosclerosis, had already been present for a considerable time The patient showed no signs of trouble, while the disturbances in the aorta, resulting from his faulty manner of living, remained fully compensated Through the unaccustomed great muscular exertion that

the patient occasionally was obliged to engage in, excessive hypertrophy of the left ventricle developed, then sudden failure in the function of this ventricle occurred, and thus a disturbance in the existing compensation was brought about. An analogy of this observed fact is that the particular symptoms of organic heart disease sometimes coincide with its course.

II. According to the accepted view, one of the first signs of beginning congestion in the venous system of the body in organic disease of the circulatory and respiratory apparatus, is a hydropic swelling of the lower extremities. I also held this view for a long time. The first case that puzzled me, I observed for nearly ten years, was a young colleague, who consulted me owing to a persistent hepatomegaly. By careful examination I found a very extensive uniform enlargement of the liver, but also insufficiency of the aortic valves. The anamnesis gave no clue, and made independent and more profound disease of the liver probable, it overshadowed the probability of a swelling from congestion of the hepatic venous system. But why did hydrops fail to appear?

After my attention was once focused in this direction, other similar cases soon came under my observation, and I gradually became convinced, that no hydrops, but swelling of the liver, was the first sign of beginning congestion of blood in the venous system of the body. Our case in comparison offers no exception to the rule, it is contrarily to be viewed as an example of usual occurrence. The old rule in regard to swelling of the liver finds new significance, in that the heart must always be considered and examined, before the diagnosis of liver disease is definitely made. In retrospect, one can definitely determine the nature of this swelling in those individuals who show no trace of hydrops, when they present uniform enlargement of the liver with smooth surfaces, but one must first undertake a careful examination of the heart.

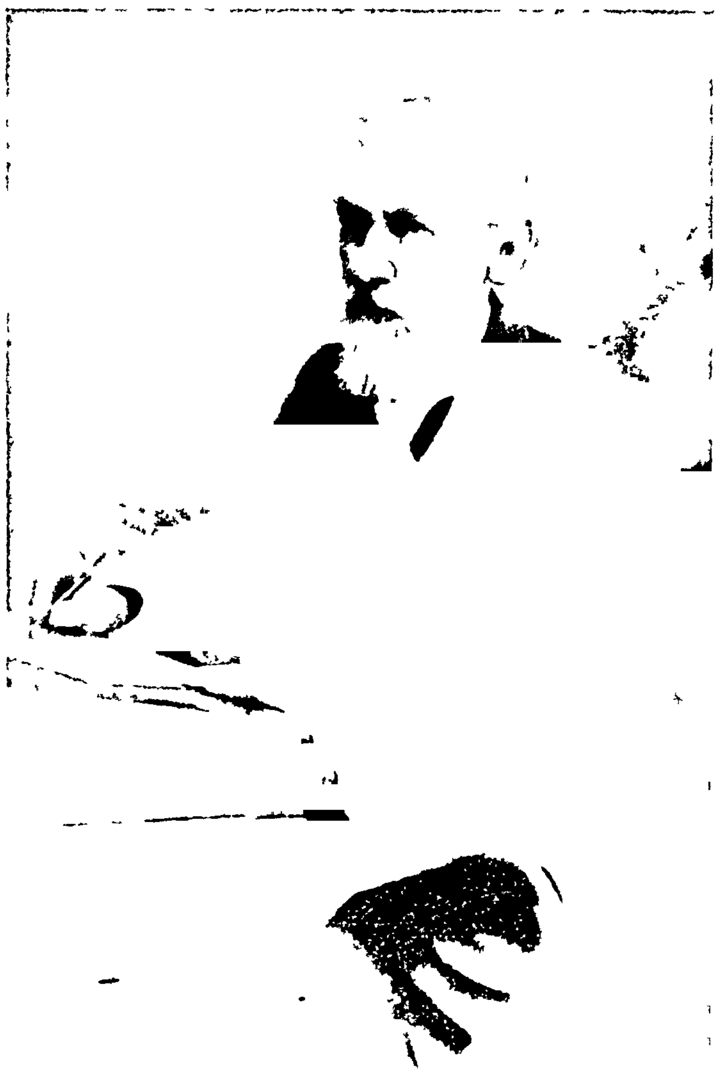
III. Owing to my absence at the autopsy, microscopic examination of the liver was neglected. I greatly regret this, as the course of this case in the final period had aroused conjecture that the patient had succumbed to acute atrophy of the liver. For the acceptance of this view, we find the following evidence: (a) the rapid diminution in the size of the liver as determined by palpation, (b) the ever-increasing icterus, (c) the peculiar form of delirium, and (d) the absence of febrile reaction. Were my conjecture correct, the case would offer a new contribution to the accepted teaching, that the acute degeneration of the parenchyma of the liver and the resulting rapid diminution in size of the liver, were accidents in the course of the various acute as well as chronic, severe as well as mild, diseases of the liver, and we would presume, that the diseases with icterus are included in this group. I recollect, moreover, of having observed various cases of heart disease, in which death occurred with phenomena similar to those of the foregoing case.

IV In conclusion, just a few words regarding our observations on pulsus alternans A review of this case from the beginning of the epicrisis must strengthen the conviction that this phenomenon was influenced, at least in part, by the digitalis I observed this phenomenon during the third period of the illness, that is, in the interval during which the patient improved while using greater quantities of digitalis, when existing failure of compensation could be recognized only by enlargement of the liver and distention of the jugular veins With the diminution in the action of the digitalis, the pulsus alternans began to disappear, and finally was only distinctly seen in the carotids But digitalis belongs, as I have shown, to those agents, which stimulate the inhibitory nervous system of the heart Following this complete consideration, we are justified in concluding the discussion of the close relationship of pulsus alternans and bigeminus

1876

WILLIAM RICHARD GOWERS

DESCRIPTION OF THE RETINAL VESSELS IN HYPERTENSION



SIR WILLIAM RICHARD GOWERS

(Courtesy Journal of Nervous and Mental Diseases)

SIR WILLIAM RICHARD GOWERS

(1845-1915)

ON MARCH 20, 1845, William Richard Gowers was born in London He received his academic training at Christ Church College School, Oxford When he was sixteen years of age, Gowers was apprenticed to a country surgeon at Coggeshall, Essex He received his medical education at University College, London, and in 1867 qualified for membership in the Royal College of Surgeons Two years later he received the degree of Bachelor of Medicine from the University of London In 1870 he received the degree of Doctor of Medicine and was awarded the gold medal for his high standing in the class

After graduation, Gowers was appointed medical registrar to the National Hospital for the Paralyzed and Epileptic He also continued his position as private secretary to Sir William Jenner, a position which he had occupied during his student days Gowers deeply appreciated his contact with Sir William Jenner, and Jenner helped Gowers in his brilliant career

In 1872, Gowers was appointed assistant physician to University College Hospital and became physician to that institution in 1883 Meanwhile, in 1873, he was appointed to the rank of assistant physician at the National Hospital for the Paralyzed and Epileptic He became a physician there in 1883 On his retirement in 1888, he was appointed consultant He also served for many years at the University Medical School as a teaching assistant and later was appointed professor of clinical medicine

Gowers was greatly interested in the diagnostic value of the ophthalmoscope and, in 1876, he published an important paper in the "British Medical Journal" entitled "The State of the Arteries in Bright's Disease" Because this article contains his classic description of the retinal vessels in the presence of hypertension, we are including it in our CARDIAC CLASSICS His more detailed work, "A Manual and Atlas of Medical Ophthalmoscopy," was published in 1879 Although the importance of the ophthalmoscope was realized by others before Gowers, this publication, which was thorough and systematic, brought ophthalmoscopy into a much wider use in general medicine than had been the case before A fourth edition of this deservedly successful book was published in 1904

In 1879, Gowers was elected a fellow of the Royal College of Physicians He had qualified for membership in that organization in 1875 In 1880, he gave the Goulstonian Lecture, choosing for his subject, "Epilepsy and Other Chronic Convulsive Diseases" This address was published in 1881

Gowers, at the beginning of his career, showed a special interest in the diseases of the nervous system In 1876 he published an essay "On 'Athetosis' and Posthemiplegic Disorders of Movement" In 1877 he wrote on "The Diagnosis and Treatment of Auditory Nerve Vertigo," and in 1878 he published an important paper on chorea, "On Some Points in the Clinical History of Chorea"

His interest in neuro-anatomy led Gowers to the discovery of the tract of fibers in the gray matter in the ventral and lateral funiculus of the spinal cord the fasciculus arteriolateralis superficialis, also known as Gowers' tract His discovery was first published in 1879 in his lecture, "The Diagnosis of Diseases of the Spinal Cord" An enlargement of this lecture as a book was published in 1880, under the same title

Gowers is also remembered for his method of estimating the percentage of hemoglobin and the number of corpuscles in the blood In December, 1878, he presented the details of his hemoglobinometer before the meeting of the Clinical Society of London His method was used until 1901, when Haldane's modification took its place

In 1885, Gowers published his famous work "Lectures on the Diagnosis of Diseases of the Brain," in which he correlated the observations of Hughlings Jackson (1834-1911), Paul Emil Flechsig (1847-1929), Eduard Hitzig (1838-1907) and David Ferrier Between 1886 and 1888 this unceasing and seemingly untiring worker published his two volumes, "A Manual of Diseases of the Nervous System" In 1899, the third edition of the work was published

Gowers published another monograph in 1892, it consisted of his Lettsomian Lectures on "Syphilis of the Nervous System" He had delivered these lectures in 1890 Many years previously, in 1879, he had delivered his first Lettsomian Lecture on syphilis of the nervous system It was Gowers' belief that, apart from embolism and injury, sudden hemiplegia occurring between the ages of twenty-five and forty-five years is very seldom the result of anything other than syphilis

In 1897, Gowers, on the occasion of the Diamond Jubilee of Queen Victoria, received the honor of knighthood Meanwhile, his health, which had become severely strained by the labor of publishing so many exhaustive volumes and the worries of a large practice, became dangerously poor To improve his health he took a long voyage and vacation to South Africa He returned to England feeling better He maintained his practice and continued to make contributions to the medical literature In 1907 he published his last important work entitled "The Border-Land of Epilepsy, Faints, Vagal Attacks, Vertigo, Migraine, Sleep Symptoms, and Their Treatment"

Sir William was the recipient of many honors He was a fellow of the Royal Society of London He received the honorary degree of Doctor of Medicine from the University of Dublin and that of Doctor of Laws from the University of Edinburgh He was elected an honorary fellow of the Royal College of Physicians of Ireland He was also an honorary member of the following societies the American Neurological Association, the Netherlands Society of Psychiatry and Neurology, the Russian Society of Medicine, the Royal Society of Science of Upsala, and the Society of International Medicine of Vienna

Sir William married the daughter of Frederick Baines of Leeds She died in 1913, leaving four children two sons and two daughters

Sir William Gowers died on May 4, 1915, after a long illness

THE STATE OF THE ARTERIES IN BRIGHT'S DISEASE*

By

W. R. GOWERS, M.D. Lond.

Assistant Physician and Assistant-Teacher of Clinical Medicine, University College Hospital

THE OPHTHALMOSCOPE is of service to the physician, among other uses, because it enables him to see the termination of a minute artery and vein, and to gain direct evidence of their condition, such as is to be obtained in no other way. When retina is free from local disease, there is no reason to believe that the retinal artery and vein differ in their condition from other arteries and veins of the same size, and, therefore, any marked change in their state, apart from cerebral or ocular disease, may be taken as evidence of a similar change throughout the vascular system.

The object of the following paper is to bring forward certain facts concerning the retinal vessels in Bright's disease, and I believe the facts warrant this conclusion, that, in chronic Bright's disease, the arteries of the retina are sometimes of normal size and sometimes very distinctly lessened in size, that this diminution in size depends upon contraction, and that this visible contraction stands, as a rule, in direct proportion to the tension of the arterial blood, as measured by the incompressibility of the radial pulse.

A few words are necessary as to the manner in which the size of the arteries is estimated. Their condition can only be seen under considerable magnifying power. Examination by the indirect method does not, as a rule, give sufficient enlargement unless a lens of very long focus be employed, as in Carter's demonstrating ophthalmoscope. Now and then, if the eye be hypermetropic and the vessels very distinct, the indirect examination with a lens of three- or four-inch focus will suffice, but as a rule, it is necessary to employ the direct method of examination. If the pupil be small, it must be dilated with atropine, since it is often necessary to trace the vessels for some distance from the disc.

There is unfortunately no method of applying any gauge to the vessels, their size must be estimated by the eye. The change in size may be judged of absolutely or by comparison with the veins. For an absolute estimate of their size, familiarity with their normal appearance under direct examination is, of course necessary. Further, as the degree of

*Brit. M. J. 2: 743-745, 1876

magnification varies with the refractive power of the eyeball, this must be allowed for. It may generally be estimated by noticing the apparent size of the disc.

The change in the size of the arteries is frequently such as to be recognised at once; there is no need for comparison with the veins. The reduction in size may be so considerable, that even the primary branches of the central artery are so small that their double contour is recognised with difficulty, and it may be unrecognisable even by direct examination, the arteries being, as in one example I have to show, visible only as lines.

In other cases where the diminution in size is slighter, it can be most conveniently estimated by comparing the arteries with the veins. The distribution of the arteries and veins corresponds approximately, not exactly. Sometimes two arteries accompany one vein, sometimes one vein corresponds with two arteries. But in each eye there are usually some single branches of arteries and veins which have an identical course and distribution, run side by side, and are available for comparison. When this is the case, it will be found that, as a rule, the width of the artery is about two-thirds or three-quarters that of the vein. When the artery bears less proportion to the vein than this, it is usually due to one of three causes: 1. General venous distension, as in cyanosis; 2. Impediment at the sclerotic ring, by which the entrance of blood into the arteries is impeded, and its exit from the veins is also hindered, in which case the arteries are narrow and the veins distended as in certain stages of optic neuritis; 3. Contraction of the arteries. In the two former cases, the veins are, of course, abnormally large, and their abnormal size is generally easy of recognition. In the latter case, the veins may be normal in size or may be smaller than natural. If they be smaller, the diminished proportionate size of the arteries is of still greater significance. It is necessary, therefore, to be familiar with the normal size of the veins, in order to estimate the size of the arteries by comparison. From their darker colour, their size is easily noted, and the size of the arteries is readily estimated by comparison.

The arteries may be of normal size upon the optic disc, and yet present very marked reduction in size on the retina, a little distance from the disc. An artery may leave the disc beside a vein to which it bears its normal proportion, and, after a little course, without giving off any visible branch, may diminish to one-half or one-third of the size of its accompanying vein.

From what has been said, it will be obvious that these changes in the relative size of the vessels possess most significance when the retina has not undergone the special changes to which it is liable in chronic Bright's disease. Exudation within the sclerotic ring, compressing the vessels, alters their relative and absolute dimension, as I have stated. This is well seen in ordinary optic neuritis. I believe that it is rare in Bright's dis-

ease for the neuritic change to be sufficient to produce this effect Certainly, in cases of albuminuric retinitis, the actual contraction may often be recognised as something quite out of proportion to the retinal change

As I have said, the rule that, when the arterial tension is increased, the retinal arteries may be seen to be contracted, is general, but not universal This is in accordance with what might be expected from the various conditions which are known to influence, on the one hand, blood-tension, and, on the other, arterial contraction Moreover, local influences may cause local modifications The most notable exceptions to the rule, which I have met with, have been in cases of local retinal disease

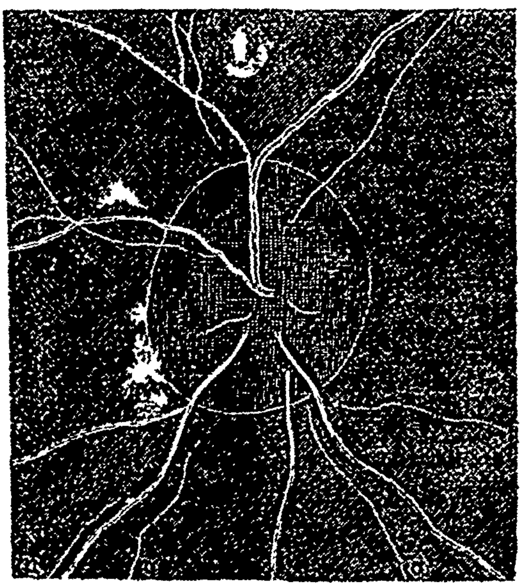


Fig 1

The following are some of the facts on which the conclusions described have been based The incompressibility of the radial pulse was employed as the estimate of arterial tension When practicable, my own estimate has been corroborated by a sphygmographic tracing, or by an independent opinion

On opposite sides of a ward in University College Hospital, there recently lay two patients (under the care of Dr Reynolds), whose cases illustrated in the most marked manner the relation of blood-tension and the state of the retinal arteries The one case was that of a man, fifty-eight years of age, whose illness had commenced gradually, with shortness of breath and weakness, two years before Slight oedema of the legs had existed for only one month before his admission His urine contained one-third of albumen, had a specific gravity of from 1 005 to 1 008

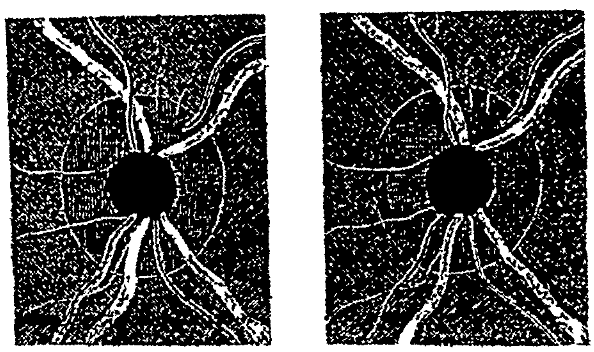
and contained numerous casts, granular and hyaline, with some degenerated epithelium, both free and within the casts His retinae were normal in appearance, the arteries of full size, presenting not the slightest evidence of contraction His pulse was full, but very soft and compressible There was no evidence of cardiac change The other case was that of a man, aged 46, whose symptoms resembled those of the first They began with shortness of breath and swelling of the legs nine months before His urine had a specific gravity of 1 007 to 1 010, and contained from one-third to one-half albumen, its quantity was from two to four pints, and it contained many casts, granular, hyaline, and epithelial His retinae presented evidence of slight disease The optic discs (Fig 1) had softened outlines, and their surface was reddish-grey, paler in the vicinity of the vessels There was little, if any swelling The veins were smaller than normal, in the left eye, one only approached the average size The arteries presented a greater reduction in size than in any case I have seen Even on direct examination they were visible only as lines, no double contour being recognisable, although they were quite distinct A few minute white dots existed in each eye near the macula lutea, and in each there were a few small extravasations The vessels were similar in the two eyes The disc in the right was a little less grey than

Fig 2

in the left Vision R = one-sixth, L, one-twentieth There was no peripheral limitation of the fields of vision The pulse was extremely hard, the artery felt like a whipcord under the finger, and was almost absolutely incompressible The strongest pressure which could be put upon it with a Marey's sphygmograph did not modify its character The accompanying tracing (Fig 2) was taken under the highest available pressure (about four hundred *grammes*)

The sepia drawing now passed round represents the fundus oculi in a case of chronic Bright's disease, the sequel of an acute attack 12 years previously, the patient having in the meantime had at least two other acute attacks The urine was loaded with albumen and contained granular and fatty casts The retina presents abundant soft-edged white areas and also many striated extravasations, most of these had appeared during the preceding ten days The disc is concealed by oedema The veins are of normal size, the artery at the papilla is rather smaller than natural, its branches being not more than half the size of the veins, but, a little distance beyond the limits of the papilla, they disappear The veins can be followed distinctly on the retina, but the arteries can only be seen as

dim lines here and there It may be suggested that this appearance is due only to the opacity of the retina, but the fact that they can be dimly seen here and there as lines suggests that their indistinctness is due in part only to the opacity of the retina, in part also to their reduction in size The greater extent of this reduction on the retina than on the disc suggests that it is not due to their obstruction at the lamina cribrosa, but to their active contraction The facts of other cases give support, I think, to this view This patient's pulse was also very hard and incompressible He insisted on going out of the hospital, but died comatose a few days afterwards A post mortem examination was obtained by Dr Burton, one of the resident assistants at the hospital The kidneys were found lessened in bulk, increased in consistence, and moderately granular on the surface The heart was hypertrophied



Figs 3 and 4

The next two sketches (Figs 3 and 4) represent the optic disc of a patient (under the care of Dr Ringer) suffering from acute Bright's disease passing into a chronic state The first was made six weeks after the onset The retina presented a few small haemorrhages, white dots around the macula lutea, with a few larger white areas The arteries and veins were normal in size, the former being just two-thirds the diameter of the latter The pulse was soft and compressible, giving no evidence of increased blood-tension When the second sketch was made, six weeks later, the patient's general condition had improved, the albumen in the urine was less, but the casts had become fatty, the retinal changes had become considerably less, the white areas had lessened The retinal arteries, however, presented distinct diminution in size compared with their previous condition The veins were apparently of the same size as when the former sketch was made, while the arteries had diminished to one-half the size of the veins The pulse also presented a marked alteration It had become distinctly harder and less compressible The change seemed to have taken place only a short time before the sketch

was made, for, on my calling the attention of the resident assistant to its character, it struck him at once as quite different from that which had been its character a short time before

I have repeatedly formed, from an inspection of the retinal vessels, an opinion as to the arterial tension, which, I afterwards found, on examining the pulse, was correct In order however, to obtain some evidence which might be without even unconscious bias, I asked my friend Dr Coupland to be good enough to examine the pulse in a series of cases of Bright's disease whilst I examined the retinal vessels Each wrote down independently the result of the examination Five cases were examined, and the results agreed in four In one case, they differed, but in this the fact that there were many retinal haemorrhages may, as I have already said, explain the absence of arterial contraction, although the pulse was hard The following are the details of these examinations

CASE I —Arteries small, about one-half the diameter of the veins, moderate contraction (retina healthy, except for a few haemorrhages, disc clear), pulse moderately tense, *i e*, moderately incompressible

CASE II —Arteries one-half the size of the veins moderate contraction (retina perfectly normal) pulse incompressible (about as the first case)

CASE III —Arteries nearly two-thirds the size of the veins, very little contraction (many extensive retinal haemorrhages), pulse markedly incompressible

CASE IV —Arteries less than half the size of the veins, great contraction (retina normal, disc clear), pulse very incompressible

CASE V —No diminution in the size of the arteries retina healthy, pulse soft and compressible

Thus, in the case in which the retinal arteries were smallest the arterial tension was greatest, that in which the arteries and the retina were normal presented no excess of arterial tension, the two others, in which there was a moderate contraction of the arteries, presented a moderate increase in the arterial tension

There is, of course, nothing new in the fact that the retinal arteries are small in Bright's disease, it has long been remarked as a common feature in albuminuric retinitis, and is shown plainly in the best illustrations of this change (as in those of Liebreich) But it is usually regarded as a consequence of the retinal change, and the points on which I would insist are that it occurs also quite independently of the retinal change, and stands commonly in direct relation to another condition—the blood-tension

It is hardly necessary for me to point out the bearing of this conclusion on the theory of Dr George Johnson, which ascribes the increased tension of the blood in Bright's disease, in part at least, to contraction of

the minute arteries It constitutes, I think, a direct proof of the correctness of the theory, which has hitherto derived its chief support from indirect inference from pathological facts If the tension of the arterial blood and the arterial contraction occur in common proportion, they must stand in a causal relation to one another But the blood-tension cannot be the cause of the arterial contraction, because it is well known from physiological experiments that the tendency of increased blood-tension is, through the depressor nerve, to cause relaxation of the arterioles But, on the other hand, as the immediate effect of contraction of the arterioles must be an increase in the arterial blood-pressure, it is reasonable to conclude that such is the sequence of events in the phenomena under consideration, that, although the two phenomena may be in part the result of a common cause (altered state of the blood), the contraction of the arteries, seen in those of the retina and inferred to exist elsewhere, is, in part at least, the cause of the increased blood-tension

The practical use of inspection of the retinal vessels is perhaps less than its pathological value, but it is, I think, considerable It is true we can generally ascertain the amount of arterial tension more readily and more surely by feeling the pulse than by looking at the retinal vessels But sometimes the incompressibility of the pulse cannot readily be estimated, on account of its smallness and the amount of subcutaneous fat or oedema In these cases, retinal inspection may be useful Moreover, as affording definite information regarding the pathological processes in different cases of Bright's disease, it will, I think, have considerable value, and some facts which have come under my observation, at present too few and isolated for more than mention, make me hope that ultimately it may help us better to distinguish between morbid states included under the term and at present imperfectly distinguished

1877

JULIUS FRIEDRICH COHNHEIM

DESCRIPTION OF PARADOXICAL EMBOLISM

Dr. Jul. Cohnheim, ord Prof

JULIUS FRIEDRICH COHNHEIM

(Courtesy Dr C W G Rohrer)

JULIUS FRIEDRICH COHNHEIM

(1839-1884)

"Without blood vessels, no inflammation is possible"

—Cohnheim

JULIUS FRIEDRICH COHNHEIM was born on July 20, 1839, in the town of Demmin, Pomerania, a province in Northern Prussia He received his gymnasium training at Prenzlau, and in 1856 began the study of medicine at the University of Berlin Later he studied at the University of Wurzburg Cohnheim pursued his studies in an ardent manner, and under the influence of Albert von Kolliker, developed into an expert histologist He remained in Wurzburg until the spring of 1860 He then studied for a few months at Greifswald in Pomerania and returned to the University of Berlin

At Berlin he passed his examination for the degree of Doctor of Medicine He then prepared his thesis under the direction of Rudolf Virchow (1821-1902), the title of his dissertation being "De pyogenesi in tunicis serosis" While working under Virchow at the Pathological Institute of the Charity Hospital in Berlin, Cohnheim became acquainted with Friedrich von Recklinghausen (1833-1910) and Edwin Klebs (1834-1913) The inspiration he received from these three men no doubt helped him in his decision to devote his life to science

Meanwhile, Cohnheim's father, who had been forced by circumstances to leave Germany for Australia, returned home suffering from a fatal illness and in July, 1862, died It became necessary for the son to contribute to the support of the family, but he spent what spare time he could in the study of normal and pathologic histology and in learning the methods of chemical investigation

In 1862, Cohnheim came under the influence of Ludwig Traube (1818-1876) He became one of Traube's students and gained not only clinical experience from this great teacher but also a knowledge of experimental physiology In appreciation, Cohnheim later dedicated his "Lectures" to Traube's memory That same year, 1862, Cohnheim passed the state examination in medicine

Cohnheim served as a surgeon in the Prussian Army in the German-Danish War of 1864 On the death of his brother, Albert, a victim of the war, Cohnheim, being the sole support of his mother, was released from military duty He then accepted a position under Virchow as an assistant in the Pathological Institute in Berlin, where he remained for seven years During this time he published many articles in Virchow's "Archiv fur experimentelle Pathologie" He developed an interest at the Institute in the genesis of inflammatory conditions and published articles on tuberculosis of the choroid, inflammation and suppuration, and on mechanical hyperemia Cohnheim was soon able to show that the pus of inflammatory conditions is derived from the blood

In 1868, Cohnheim, who then was only twenty-eight years of age, was offered the newly established chair of pathology and pathologic anatomy at the University of Amsterdam This he declined in favor of the chair of pathology at the University of Kiel At Kiel, Cohnheim continued the work on the pathologic aspects of the

circulatory system which he had begun in Berlin under Virchow He began the study of embolism During the summer of 1869 he studied disturbances of the circulatory system under Karl Ludwig (1816-1895)

On May 20, 1872, his marriage to Martha Lewald took place Three sons were born to them In the fall of 1872, Cohnheim accepted a call to the medical department of the University of Breslau At Breslau he suffered from attacks of gout, the symptoms of which he had noted while he was at Kiel He spent the winter of 1873 1874 at Montreux This appeared to help him

At Breslau, Cohnheim founded a pathologic institute, which was planned to be similar to the Pathological Institute at Berlin

In 1875 Cohnheim met Robert Koch (1843-1910) Koch was then a country practitioner in Wollstein and he came to Breslau to submit to Ferdinand Julius Cohn (1828-1898) his cultivations of the anthrax bacillus On seeing these remarkable cultures, Cohn asked that someone from the Institute view them Cohnheim did so and said of Koch's work, according to McKee, "I regard this as the greatest discovery in this domain, and believe that Koch will again surprise and put us all to shame by further discoveries" Cohnheim lived to see Koch's discoveries of the *Mycobacterium tuberculosis and the vibrio which causes cholera*

Cohnheim published the first volume of his "Lectures on General Pathology" in 1877 We have chosen to reproduce from McKee's translation, published in 1889, *Cohnheim's famous description of paradoxical embolism*

In 1878, Cohnheim was called to the University of Leipzig to become ordinary professor of general pathology and pathologic anatomy There the second volume of *his "Lectures" was written and he undertook new investigations concerning the* pathology of the circulation, for which he is renowned

In the summer of 1878 his attacks of gout became more frequent He spent the summer at Carlsbad, hoping *to effect a cure, but the attacks persisted and in 1883* he was forced to give up his lectures His strength gradually failed, and he died on August 15, 1884 His friends and admirers erected a monument to his honor at Leipzig

LECTURES

ON

GENERAL PATHOLOGY.

A HANDBOOK FOR PRACTITIONERS AND STUDENTS

BY

JULIUS COHNHEIM,

ORDINARY PROFESSOR OF GENERAL PATHOLOGY AND PATHOLOGICAL ANATOMY IN THE UNIVERSITY OF LEIPSIC

TRANSLATED FROM THE SECOND GERMAN EDITION

BY

ALEXANDER B McKEE, M B DUB,

CURATOR OF THE MUSEUMS ROYAL COLLEGE OF SURGEONS IN IRELAND

WITH MEMOIR BY THE TRANSLATOR.

SECTION I

THE PATHOLOGY OF THE CIRCULATION

LONDON

THE NEW SYDENHAM SOCIETY.

1889

(Courtesy Dr C W G Rohrer)

THE PATHOLOGY OF THE CIRCULATION

By

JULIUS COHNHEIM

Ordinary Professor of General Pathology and Pathological Anatomy in the University of Leipsig

SECTION I, CHAPTER IV

THROMBOSIS AND EMBOLISM

But the locality to which the detached thrombi are transported is determined by anatomical conditions alone Coagula from the venous side of the vascular system, *i e*, from the veins and the right heart, arrive in the *pulmonary arteries,* those from the arterial side, *i e*, from the left heart, the systemic arteries, and the pulmonary veins are conveyed into the *aortic system,* while those from the portal tributaries reach the *branches of the portal vein* in the interior of the liver Not only are these general fundamental rules of transport throughout the vascular system prescribed by anatomical structure, but the special paths within any given portion of the circulatory mechanism are also so prescribed The cause determining the entrance of the embolus into one artery rather than another can, at least in the case of larger plugs, be as a rule satisfactorily determined, and is to be sought in the direction of the embolized vessel with respect to the principal trunk, *i e*, in the angle at which it is given off, or in the relative calibre of the various lateral branches, or in some similar circumstance Where the transport of the embolus appears to take place in opposition to anatomical laws, there are usually, as though to prove the rule, anomalies in the distribution of the vessels or in the heart Thus I had quite lately an opportunity of observing a case of recent fatal embolism of one of the mid cerebrals in a woman thirty-five years of age, where the valves of the heart, aorta ascendens, in short all the arteries from which an embolus might have been conveyed, were absolutely intact, while on the other hand an extensive thrombosis had occurred in the veins of the lower extremity I had not, as you may suppose, at first the remotest idea of connecting the two conditions, till on more carefully inspecting the heart, I discovered a *foramen ovale* so

*The original German publication appeared in 1877 We are reprinting from the English translation published in 1889 *Lectures on General Pathology* translated by Alexander B McKee London The New Sydenham Society 1889 Section I, pp 182-184 —F A W and T E K 1940

large that I could easily pass three fingers through it. I could not any longer reject the possibility that here a thrombus carried off from the *v. femoralis* had on its way through the heart passed from the right into the left auricle and thence into the mid. cerebral.* But the more one sees how perfectly the anatomical conditions are maintained in the course of such events, the more difficult, it seems to me, will it be to make up one's mind to indorse the opinion of certain authors that in cases, it is true of rare occurrence, emboli may be transported against the blood-stream, as for example, out of the *vena cava superior* into the hepatic vein.† It is an old experience and capable of verification at any moment that particles, say, of cinnabar, granules of mercury, or even plugs of wax may be very readily driven by the positive pressure of an injection-syringe from the *v. jugularis* into the hepatic veins, but this proves nothing with respect to the ordinary circulation, and I should, for my part, regard such an occurrence as very improbable, so long at least as the blood-stream through the portal vein is unimpeded.

*Litten (Virch. A., IXXX, p. 381) describes a similar case, in which the *foramen ovale* being patent, a thrombosis of the right auricle was believed to have formed the starting-point for repeated embolism throughout the systemic vessels.

†Heller, D., Archiv f. klin. Med., vii, p. 127; Wagner, Allg. Path., p. 281.

1879

HENRI ROGER

DESCRIPTION OF THE MURMUR OF PATENCY OF THE INTERVENTRICULAR SEPTUM

HENRI LOUIS ROGER

(Courtesy Faculty of Medicine, University of Paris)

HENRI LOUIS ROGER

(1809-1891)

HENRI ROGER was born at Paris on June 15, 1809 Following the example of two friends of the family, Drs Guersant and Blache, he decided to become a physician In 1833 he served an internship at the hospitals of Paris, and in 1847 he became physician to the hospitals of Paris and that same year he qualified for membership in the Faculté de Médecine He served as physician to the Hospital for Sick Children from 1853 to 1874 He also was associated for twenty-two years as physician to the Sèvres Street Hospital and from 1862 until 1874 he was in charge of the Clinic in Paris

Roger occupied a prominent position in the Association Générale des Médecins de France He was president of the Société Centrale in 1872 In 1876 he was elected president of the Association Générale des Médecins de France and was constantly re-elected to this office until his death on November 15, 1891

In 1839, the Societé de Médecine et de Chirurgie de Bordeaux offered a prize for the best paper on the subject, "To determine what progress has been made in diagnosis and treatment of diseases, particularly those of the lungs, heart, and great vessels, by means of auscultation, either mediate or immediate" Barth and Roger, who at that time were interns, submitted a paper for the award which had for its motto, "If medicine is the most beautiful of the sciences, then auscultation is the most beautiful discovery of modern times" Although this paper did not receive the prize, which was awarded to Peyraud of Lyon, it did receive an honorable mention It was the basis of a more extensive work by Barth and Roger on auscultation first published in 1841 This publication, greeted with enthusiasm by the medical profession, went through several editions and was translated into a number of foreign languages including the Scandinavian

Roger's keen interest in auscultation combined with his skill as a pediatrician led him to discover an important anomaly of the septum, simple interventricular communication, later known as "Roger's disease" In 1861, in performing a necropsy on the body of a young boy about twelve years of age, he found a malformation of the heart which consisted of failure of occlusion of the interventricular septum in its upper portion, without concomitant stenosis of the pulmonary artery Necropsy also showed that the communication between the two ventricles would occur without cyanosis After having listened to the heart sounds of thousands of children, Roger, with the aid of this pathologic discovery, was able to demonstrate that this lesion was characterized by the presence of a thrill and systolic murmur situated at the middle of the heart The condition was not accompanied by any functional symptoms, cyanosis in particular and dyspnea being completely absent Roger was also able to show that this congenital defect could exist without alteration of general health

In 1879, Roger, after demonstrating this lesion in several instances, presented his observations to the Academy of Medicine It is our privilege to present to our readers, in translation, Roger's observations entitled "Clinical Researches on the Congenital Communication of the Two Sides of the Hearts, by Failure of Occlusion of the Interventricular Septum"

CLINICAL RESEARCHES ON THE CONGENITAL COMMUNICATION OF THE TWO SIDES OF THE HEARTS, BY FAILURE OF OCCLUSION OF THE INTERVENTRICULAR SEPTUM

By

HENRI ROGER

AMONG the congenital defects of the heart compatible with life and perhaps a long one, one of the most frequent which I have encountered (relatively frequent, absolutely rare) is the *communication between the two ventricles because of failure of occlusion of the interventricular septum in its upper portion*

Of these cases (of which I have seen about a dozen), some *have cyanosis* and some *do not*, and as there should be, in all cases, as a result of this communication, a mixture of the two bloods, I have concluded with Louis and Gintrac, who have best established this proposition, that the *morbus ceruleus* does not arise from *the combination of arterial and venous blood*, but that it is almost always attributable to a concomitant and likewise congenital lesion, *stenosis of the pulmonary artery*, the effect of this stenosis being to impede and prevent the arrival of the blood in the lungs and consequently the decrease of oxygenation

Leaving aside the cases of cyanosis where the diagnosis should spring up before the eyes, so to speak, but which are nevertheless complex and where the anatomical conditions do not always reveal themselves to the listener through abnormal sounds, I am going to speak here only of simple cases, where there is a *communication of the two ventricles without morbus ceruleus*

This cardiac anomaly has no objective symptom which the eye can recognize, it is dependent almost entirely on auscultation After having listened to thousands of children during forty years of special studies, and because of incessantly repeated stethoscopic examinations, I am able, with the control of pathologic anatomy, to separate this anomaly of the heart from other *malformations* and *diseases*, to establish its distinct clinical existence, and to make of the *murmur* which characterizes it, a pathognomonic sign

It was many years ago in village practice that I first recognized the peculiar facts of auscultation, when I was called to consult about the

*Roger, Henri Recherches cliniques sur la communication congenitale des deux coeurs par inocclusion du septum interventriculaire Bulletin de l'Académie de Médecine 2me série Tome VIII, 1879 pp 1074-1094 Translated by J P Wozencraft M D, Rochester, Minn

health of children, either for a recognized or suspected affection of the heart or for some other disease (not connected with the heart), I found, *among such children, almost all of them quite young,* a murmur of remarkable intensity, but with other characteristics which I thought unusual, what surprised me was that the murmur was almost the only sign of cardiopathy, and that it was accompanied by no other physical signs (save only the *purring thrill*), nor by any functional troubles indicating any lesion of the orifices or even of any signs of alteration of blood (since chloranemia is not at all a disease of early infancy, and the abnormal sounds heard in the very young are almost always on an organic basis)

What surprised me greatly while I had an opportunity to attend these children, to see them again, and to listen again at intervals more or less wide, was to find the same murmur after months or years with the same characteristics, without any appreciable alteration, without new physical signs, without alteration of general health, and *without cyanosis*

I am going to cite three cases of these of which I have notes or precise memoranda The first concerns a little boy of four months (Eugene L) whom I saw several times and in whom I found at each examination the characteristic murmur unchanged, as was his general condition I did not see him after the age of five years, his father, a physician, had set up practice in the province In the second case I continued to see the little patient (Henry de B) from the age of six months to his eighth year, when he was removed to Havre by his father, a customs collector Several examinations, repeated at intervals of several years, showed identical results, an abnormal sound with a thrill which persisted without change, unaccompanied by any other sign of heart disease

I have had the opportunity of observing a third patient during a much longer period of time, together with Blache he was a Grammont-Caderousse, elder brother of the young duke who died of consumption and who was well known during his life for his eccentricities and after his death by the lawsuit of a physician, equally well known, to whom he had left his fortune In this case, as in the others, I found a cardiac murmur having the aforementioned characteristics, and I found this murmur at each examination, persistent and unchanging, without visible damage to the organism After about fifteen years I lost sight of this young man (the diseases of his youth required a specialist other than a pediatrician), until I read in the papers that a large steamer which was taking him to America had sunk in mid-ocean Later I learned from the family that the elder brother had not taken any better care of himself than had the younger, and that, although he was not robust (the mother had died of a tuberculous pleurisy), he had never appeared to suffer from organic heart disease

These extraordinary cases, presented to my observation at long intervals, are difficult to interpret I asked myself if this permanent *murmur* in the precordial region indicated an *endocarditis* But if the abnormal

sound resulted from an alteration of the valves and orifices, why should it not show modifications of intensity and quality according to the constant progress of the affection, and further, why did not serious functional disorders finally manifest themselves?

On the other hand, the prolonged innocuousness of this supposed endocarditis was astonishing to me, and I asked myself rather if these little patients did not have a congenital anomaly of the circulation if the murmur did not indicate a *communication between the two hearts* and if it was not produced by the passage of the column of blood across the orifice of the communication [1] But if this supposition was correct, how did it happen that there was no *cyanosis* since it is generally admitted clinically that cyanosis is always the result of *communication between the two hearts and a result of the mixture of the two bloods?*

It was in the amphitheatre of L'Hôpital des Enfants, about 1861, that I saw the light and that I discovered the reason for these obscurities and apparent contradictions, in a young boy of 12 years, dead as the result of a comminuted fracture, I found at necropsy a *malformation of the heart,* which consisted of failure of occlusion of the interventricular septum in its upper portion, without concomitant stenosis of the pulmonary artery, in spite of the mixture of the two bloods which had resulted, neither the skin nor the tissues had been of blue color during life It goes without saying that this malformation had been completely unrecognized by an entirely pardonable omission on a surgical service, the failure to listen to the heart

Clinical circumstances had given me extraordinary facts, a chance occurrence of pathologic anatomy had offered the explanation This necropsy, which showed that *communication between the two hearts could occur without cyanosis,* supplied the reasons for the facts that were little understandable until then, I did not doubt but what my former observations were of this same malformation which was shown to me so fortunately on the cadaver, and forthwith, applying this gift of morbid anatomy, I concluded that the *cardiac murmur, with particular characters* which I had discovered in my little patients, was a *pathognomonic sign* of the malformation

It is necessary to explain in the same fashion cases similar to mine which clinicians have been able to find in children who were not cyanotic they are mistaken as to the pathologic significance of the murmur heard,[2] attempting to connect them with an organic alteration of the orifices, it is not at all possible to throw these aside at the first observation, and even if they should be considered an anatomic anomaly, there is greater likelihood that it might be an acquired lesion, common and well known, rather than a congenital one, slightly known and wholly exceptional

Thus are explained many other cases recalled further when I have been able to attend the patients during a long period of time and which have

[1]See Footnote 1 p 633
[2]See Footnote 2, p 634

furnished me proof that the failure of occlusion of the interventricular septum, when it is simple and not complicated by an anomaly, such as stenosis of the pulmonary artery, could exist and persist without compromising life or health

Examples of the developmental anomaly, which I have been able to demonstrate as a clinical reality, are rare and are not found except by accident It is probable, however, that (as is the case with newly described pathologic states) they will be found more numerous when, better known, they are studied with more care and observed more attentively This was brought to my attention by the following

On the thirtieth of last July a young man of seventeen came to ask for a certificate of physical fitness for employment as a postman, he was of small stature but of robust appearance and good health, he stated that he had never had a serious illness, he was not subject to bronchitis, palpitations nor breathlessness, he maintained that he could run as well as his friends *He did not have cyanosis* and his color was ruddy and animated

On applying the ear to the precordial region I heard forthwith a *harsh murmur* covering entirely the tic-tac of the heart, which could not be heard at any point This murmur had its maximum intensity between the nipple and the sternum, somewhat stronger near the sternum, and from this center it was transmitted in all directions equally, diminishing by degrees according to the distance, and there was coincidentally a great *purring thrill* Vertical dullness did not exceed the established limits by more than a centimeter (from the third rib to the fifth interspace) The cardiac impulse was strong and most clearly visible at the center noted, near the costochondral junction and not at the apex

If we compare the symptoms of cardiopathy found in this young man with those which I have pointed out in the preceding observations and the general picture which I now portray, does not the diagnosis of *failure of occlusion of the interventricular* septum follow clearly from the comparison?

Nevertheless, this type of communication of the two hearts may not ordinarily be recognised at first, in the majority of cases the diagnosis is made by stages, and it may not be until after several observations that the observer, bearing in mind the true significance of the murmur, will be able to attribute it definitely to the malformation

Let us review and further stress the *diagnostic data* furnished by *auscultation*, by the *comparison of local and general symptoms*, and also by *accessory considerations*

These latter are by no means the least important for diagnosis, let us suppose, for example, that a physician has found a *cardiac murmur in an infant at the breast*, he will be in a great difficulty in determining its *clinical significance*—Should he call it an *endocarditis*, and consequently

an organic murmur? But endocarditis, primary or secondary (a large source of abnormal sounds), is not a disease of the first years, and I do not recall having seen it before the second year I have published a case of rheumatic endocarditis in a child of three years, and recently I have seen a report by Dr Lecorche of endocarditis "a frigore" in a little boy of thirty-three months As for torticollis and scarlatinal rheumatism, with which very young subjects may be afflicted, they do not have the tendency to involve the heart —Should he call it an *inorganic murmur?* But *anemia,* which numerous causes (rachitis, tuberculosis, inanition) make quite common *at the beginning of life, is never manifested by a cardiac murmur*

Thus, on the sole consideration of the *age* of the patient, the significance of the murmur is already presumptive, and one may announce *a priori* as a rule almost certain, that *a murmur in a suckling infant will be most often the result of an anomaly of the central circulation rather than of a disease*

But it is *auscultation* which gives certainty to the facts which I have reported, and which I summarize from the text of my observations, the physical *signs* and above all *stethoscopic* signs, upon which I base the *diagnosis of patent ventricular septum*

I have said that the sound, or rather murmur, indicating this communication of the two ventricles has *particular characteristics* —It is generally remarkably *intense,* its *maximum* point is not at the apex (as in alterations of the auriculoventricular orifices), nor at the right base (as in stenosis of the aorta), nor at the left base (as in stenosis of the pulmonary artery), *this maximum is in the superior third of the precordial region,* and it is *median,* as is the interventricular septum itself —It is *single* and greatly prolonged, beginning with systole and always covering the two normal sounds (a prolongation not usual with the murmurs of endocarditis), it replaces, or at least masks the natural tic-tac —It is *stationary,* without transmission along the great vessels, whereas this transmission is often produced by pathologic murmurs arising from stenosis of the arterial orifices —From the central point where it is at its greatest, it extends in every direction equally, and decreases in every direction, with the same regularity, according to the distance of the ear from its center —The murmur is *coincident* with a strong impulse of the total mass of the heart, *without any very appreciable impact* at the apex, and with a *purring thrill,* of wide extent, which is in exact correlation with it

With these *positive signs,* the murmur should be distinguished from that which results from a lesion of the cardiac orifices The other signs, which are *negative,* should facilitate further the *differential diagnosis* thus, in malformations of the heart, variations are not seen which diseases show, variations in cardiac dullness, more or less spread over the precordium (with or without bulging), according to variations in the degree of energy of the beat and impact with which the region is displaced, variations in the

intensity of tactile vibrations which are equally changeable, these differences are in accord with the various organic lesions of the heart and pericardium

Indeed, a special symptomatology should correspond to these distinct anatomo-pathologic conditions in *developmental failure* the lesion is single, identical with itself, changing slightly in the course of years, and the physical signs of it are few, permanent and, so to speak, unchangeable (with accompaniment of only slight difficulties of function which vary little) On the contrary, in *heart disease,* the lesions are multiple, inconstant, subject to change more or less rapid, these diseases (pericarditis, endocarditis and aneurysms) progress by periods, they have an acute stage, then a chronic one with or without exacerbations Local and general symptoms of this complex and mobile picture show these successive phases and vary incessantly with the morbid process

It is known that heart diseases, notably endocarditis, are very often *latent* in the first stage, the difficulties of circulation and respiration are often so slight at the beginning that they escape maternal vigilance, many children afflicted with chronic endocarditis with hypertrophy run, jump and play as if they were in perfect health, and without complaining afterward of palpitation or breathlessness, which, however, are actually present It is the same in nearly all cases of patency of the interventricular septum Thus, usually the physician is not consulted until more or less later, and on the occasion of some accidental illness If he does not examine all of the systems, whether they are troubled or not (the most attentive pediatrician has the greater chance of being the best), if, impressed by the idea of the rarity of cardiac pathology in the early years of life, he neglects auscultation of the precordial area, he will necessarily pass by the true diagnosis and he will be ignorant of the murmur which would have revealed the cardiac lesion If, on the contrary, he remembers to use his ear, he will immediately perceive the abnormal sound which will show at least that the diseased organ is the heart Possibly he will mistake the significance of this murmur and will believe that an endocarditis is present (this is the only error that should be possible), but such an error, until then inevitable, he will correct in good time by carefully comparing the characteristics which differentiate the murmur of patent interventricular septum and murmurs belonging to the orifices, characteristics which I have recognized and of which I have shown the clinical value

The precision of diagnosis, of interventricular patency which I believe I have made hereafter easy, is concerned especially with *prognosis,* and it is interesting to know that an infant, especially if he is very young, has a *congenital anomaly* of the heart, through which life will not be directly endangered, and not an *organic disease* Without any doubt, communication of the two ventricles is a serious anomaly of circulation and of aeration congenital and consequently irreparable, it has its own seriousness

Nevertheless, this seriousness is less than that of phlegmasias of the pericardium and endocardium, where the damage is double, by accidents perhaps quickly fatal in the acute stage, and by those of the chronic period where progress is disastrous and the outcome always fatal. Endocarditis, less serious than pericarditis, is curable in theory and it sometimes heals, but in fact when the resolution of the inflammatory products delays more than one or two years, when secondary lesions of the orifices are advanced, healing is not to be hoped for; one may beguile himself in vain that with the changes brought about in the organism by the climacteric period these organic changes will correct themselves: time, far from diminishing the disease, makes it worse, and *it is known that the nature of these organic* affections is unceasing progression with lesions which were at first local becoming generalized. It is a pathologic circle that widens more and more.

In these diseases the peril is at least as great as in simple malformations and much more acute. I have seen a few infants recover from an acute endocarditis, but I have never seen any stricken with chronic endocarditis who reached mature years. The chances of survival are actually greater in patients with an anomaly than in those with heart disease: with the latter, young patients cannot hope to live more than an average of ten years, with the former, the average is two or three times as long.

Several subjects whom I have been able to observe, I have attended for periods of *five, twelve and fifteen years*; these children have grown like others, not one has died prematurely, and except for a tendency to pulmonary catarrh the general health has not been compromised in a single one by reason of the cardiac malformation. It is certain that they should have lived well beyond the time when I last saw them, because at the time of the last visit they appeared stronger and in better health than at the first.—The young girl whose history was reported in our *Traité d'auscultation* was sixteen years old and was in good health.—The boy whom I saw recently was *seventeen* and he appeared very capable of carrying out the laborious work of postman.

Finally, now for some twenty years, I have occasionally visited as physician a woman whose children I attended from early ages and who contracted scarlet fever from one of her children: she had always been in excellent health and had never complained of cardiac difficulties. On auscultation I was greatly surprised to hear a murmur, with characteristics which struck me as very peculiar, which I thought immediately was the result of a congenital anomaly. I asked her if physicians had ever found *anything wrong with her heart*, and she told me that Guersant the Elder (the famous pediatrician, who was my first master in infantile pathology) had recognized in her a few days after birth, a cardiac malformation.—This woman has now passed her fiftieth year; her health continues to be perfect and she is the mother of four children. I went to see her again after several months to pay my respects on the marriage of her elder

daughter, and I asked permission to listen again (by virtue of her being such an unusual woman) and I found again in the precordial region the same murmur as previously, with all of the characteristics of the *murmur of patent interventricular septum*

It can be understood that there is no direct treatment for the cardiac malformation the pathologic state of the heart existing before birth and consisting of an arrest of development is not susceptible to favorable changes, either by spontaneous evolution or by medical or surgical intervention, there is then, in these unavoidable conditions, nothing to attain, for the present or in the future, by medicine or reparative surgery I say only, to lighten the severity of this conclusion, that the congenital lesion itself does not, like a disease, progress, if one may not hope for the diminution of a disease, one does not at least have to fear an augmentation, and danger will not come unless morbid changes supervene and complications occur

The prevention of complications is by means of *hygiene*, it is this which will furnish the means of attenuating the effects of the anomaly in the circulation, and of prolonging life, not only to the average limit but even beyond Children who have been found to have patent interventricular septum should be, as the saying goes *mollycoddled*, it is necessary to preserve them with vigilant care from *cold*, which engenders catarrh and rheumatic affections, and to prevent diseases of the respiratory tract which are, with them, more serious, disorders of the respiratory function aggravating the circulatory disorder and vice-versa In the second period of childhood as well as in the first, it is equally necessary to observe precautions to prevent as far as possible any broncho-pulmonary accident

It is necessary to follow this preventive system for a long time and perhaps never to depart from it, because these patients, whether adolescents or adults, cannot live as does everyone else and do with impunity what normal and healthy people can do without damage

Everything which strains the heart, accelerates the beat, augments the activity and tends, after a time, to produce secondary hypertrophy, should be avoided These recommendations are not addressed, it should be well understood, to the very young While they are in the cradle or the maternal knees and arms, they are preserved by their sedentary life from circulatory difficulties and for this reason from hypertrophy, and with them, in the earliest years, the cardiac anomaly is wholly *latent* (except to the physician) For older children it is necessary to interdict excessive play, violent exercises and above all, gymnastics (beloved by mothers who wish to make little athletes of their children) The same interdictions hold for adolescents, and for those who have arrived at adult age one should give the advice (easy to give, difficult to follow and almost always unheeded) to abstain from all excesses, one should endeavor to make them understand that their health is their reward, and that prolongation of life

will recompense their moderation this is the hygienic precept of the poet—
He who would ride far should spare his horse."

In a word, although prudence is necessary in each case, there is no advantage in treating this *malformation of the heart, with regard to hygiene*, as if it were a disease, properly speaking.

Formerly, consultations for organic affections of the heart ended with this last prescription "Avoid emotions of mental distress."—This traditional formula, even more used today, has always seemed to me naive as well as banal strokes of bad fortune, painful emotions, deep chagrin, come to everyone without being sought the more excitable the nervous system, the more it is disturbed. Whether he is sick or well, man is not the master of his emotions because he does not command the pleasant or unpleasant events of which he receives the impression he is not able to *avoid* one or another at his will, and least of all, by means of a medical prescription.

The *service* which an accurate diagnosis of patent interventricular septum renders the patient affected is none the less real because it is *indirect*. As much as it is necessary that the practitioner should guide heart disease *in infants and adults with energetic and persevering treatments*, it is equally useless *and even harmful to give such medication for cardiac malformations*, as much as action is necessary in the first case, by so much is inaction better in the second.

Actually, all of the resources of therapeutics (and they are unfortunately quite few and altogether impotent) should be used to combat cardiac phlegmasias at their beginnings (local bleeding, resolving and soothing applications, revulsives, vesicants, etc.), to moderate them and to obtain a cure, which is, however, unusual. *And later, in spite of the development of organic lesions, in spite of the difficulty, not to say the impossibility*, of resolving or impeding secondary degeneration, it is necessary, by rational medication incessantly continued and varied, *and otherwise, to* meet therapeutic indications shown by the local lesion, by the general difficulties which arise from it and by the complications which take place on every side. But what is the opportunity, what is the utility of a similar treatment given for a malformation which is known to be irreparable?

With *digitalis*, for example, what good can be expected from this medicament so valuable in the course of cardiac affections and so necessary in exacerbations, of this indispensable medicament which practitioners (even those who deny its good effects on theoretical grounds) will not be *without? Limited in its salutary action, it is only positively* useful in certain phases of disease, *it is incapable of changing a permanent anatomo*-pathologic state like malformation, with this state that continues indefinitely, one should have a remedy the effectiveness of which should be equally inexhaustible but it is known that digitalis, effective as it is when given temporarily, becomes harmful when its use is prolonged, the therapeutic effects of the herb being replaced by the toxic ones.

What should we say of the application of cautery to the precordial region? Formerly it was the classical treatment for cardiac affections, many years ago, during my internship in the Hôtel-Dieu, I recall that the famous Récamier (an intrepid and inventive therapeutist, who was never disarmed or discouraged by the most desperate case) had the custom of prescribing in such a case, four cauterizations of the precordial region, in order, he said, to circumvent the disease on all sides Did the disease thus attacked yield? The majority of physicians of that time cherished the illusion, and their convictions were little disturbed by contradictions of the autopsy

When patent interventricular septum is recognized such *energetic* medication is not appropriate, it is absolutely proscribed by virtue of the diagnosis It is irrational to use cautery, vesicants or any other cardiac remedy to treat an incurable congenital defect With these irrevocable conditions, what should the physician do? He should attempt to retard, by means of the resources of hygiene, the unfortunate effects of nature's error and to attenuate the subsequent influence of the local lesion on the general health; his power does not extend very far and the hope of cure is impossible, he should not torment and fatigue the organism by disquieting medication, inefficacious as well as irrational and finally harmful It is said with reason that the progress of diagnosis contributes to that of treatment, here, against an insurmountable obstacle, progress is at a standstill, the obligation of the therapeutist is to forbear and to efface himself before the hygienist An exaggerated medication surely has more dangers than pure expectation *primo non nocere*, is the adage of ancient medicine and it will be the last conclusion of this work

Footnotes

(1) By stating several facts known to science, I have completed, *a posteriori*, my response to appropriate observations presented to me after reading this paper

In patency of the interventricular septum, I do not see *any other cause for the murmur* than the *flow of blood* from left to right *across the communication* The mixture of arterial and venous blood which must take place is scarcely contestable, when we recall the differences of pressure which exist, according to the experiments of Marey, between the two ventricles, the force of contraction of the left is equal to 128 millimeters of mercury, that of the right, only 25 In the cases in which stenosis of the pulmonary artery is present, the right ventricle becomes hypertrophied and the left atrophied, and it is possible that with the relationships being reversed, blood should cross the abnormal opening in the septum with the murmur from right to left But with these complex conditions it is difficult to know what the acoustic phenomena should be and what their mechanism is without considering that stenosis of the pulmonary artery gives rise to a murmur which adds peculiarly to the difficulties of a precise and complete diagnosis

Am I deceived of the significance of the *murmur* and of its clinical value? Could it be the sign of *persistence of the foramen ovale* or of the *ductus arteriosus* and not of interventricular patency? This opinion does not appear at all tenable to me, the contraction of the auricles being impotent to force the blood from one auricular cavity to the other with enough force to produce a murmur Moreover, the isolated existence of

one or the other of these congenital abnormalities is quite rare, and they are generally surpassed by much more serious congenital lesions with more accentuated signs, thus stenosis of the pulmonary artery, with the cyanosis which it produces, is, so to speak, the forced accompaniment of patent foramen ovale, since this patency is almost always the effect of the arterial obstruction In about thirty of the cases of patent foramen ovale cited by Peacock in his scholarly work *(On Malformations of the Human Heart)* there were not more than four or five in which this malformation *appeared* to be simple, I say *appeared*, because in the majority there was cyanosis, which means the likelihood of some other concomitant cardiac lesion

Of all of these cases, only one (observed by the physician of St Thomas's Hospital) is of some value from the point of view of *auscultation* in a young girl who died of consumption after having showed also symptoms of heart disease, there were found at necropsy, in addition to tuberculous changes, a mitral stenosis with slight hypertrophy of the right ventricle, and a *persistence of the foramen ovale,* the opening about the size of a shilling During life she had had neither *maladie bleue* nor any murmur anywhere in the precordial region

Dr Ernest Labbee presented to the Anatomical Society in 1865 a heart in which *persistence of the foramen ovale* had been shown, the patient afflicted with this slight malformation was an old man who had never had any cardiac difficulties and in whom auscultation, frequently done, did not reveal any *murmur* or any other alteration of the normal heart sounds

Dr Duroziez, an exact and conscientious observer who has consecrated long years to the special study of vascular sounds and cardiac murmurs was asked if persistence of the foramen ovale could give rise to abnormal sounds he said that he had never found an example and that the rare observations which had been cited did not appear demonstrative to him

As for me, I have found only *one example* in the amphitheatre of l Hôpital des Enfants, of *persistence of the foramen ovale* in the absence of any other cardiac lesion, during life this anomaly had not given rise to any evidence of circulatory functional difficulty nor to any auscultatory sign

I oppose the same considerations and the same facts to the theory which seeks to assign the above described *murmur* to an origin in the *ductus arteriosus* and to the passage of blood through this patent duct for a physical cause Here, further, positive observations are absolutely wanting, and no mention is made of a murmur indicating this malformation in any of the numerous cases reported by the writers (*vid* Peacock, *loc cit*, and the admirable *Traité clinique* of Bouillaud)

There are, on the contrary, *two negative observations* reported to the Biological Society by Luys and by Duroziez The first concerns a woman, fifty eight years of age, in whom was found at autopsy a direct communication between the aorta and the pulmonary artery by means of a *persistent ductus arteriosus* which would admit the tip of the ring finger, hypertrophy of the right ventricle was also present *No abnormal sound had been heard in the region of the heart* The second case, a man forty years of age, was found to have an hypertrophy of the right ventricle with dilatation of the pulmonary artery and relative atrophy of the left ventricle *the ductus arteriosus was persistent* Auscultation, practiced frequently, *did not reveal any cardiac murmur*

Without denying the possibility of the existence of a cardiac murmur in a case of patency of the foramen ovale or the ductus arteriosus, I consequently affirm the diagnostic value of the *murmur* which is *almost always* the sign of *patency of the interventricular septum*

(2) In the eighth edition (1874) of *Traité d'auscultation,* there is recorded in a note (p 476) an observation collected by my collaborator and perpetual friend, M Barth, where he caught a glimpse of what I see clearly today "In a young girl of sixteen

years, there was found over the heart an intense sonorous *murmur* with markedly pronounced *purring thrill*, the murmur had its maximum at the level of the *bifurcation of the pulmonary artery* and was diminished according to the distance from this point What might be the cause? Could it be the *persistence of the ductus arteriosus* or rather a communication of the two hearts because of patency of the interventricular septum? The lesion appeared to us to be congenital The girl was in good health otherwise and could sing without difficulty *we did not note cyanosis* ''

Conclusions

1 A *developmental defect of the heart occurs* from which *cyanosis* does not ensue in spite of the fact that a communication exists between the cavities of the two ventricles and in spite of the fact that admixture of venous blood and arterial blood occurs This congenital defect, which is even compatible with a long life, is a simple one, without the association of congenital pulmonary stenosis It comprises a defect in the interventricular septum

2 It is necessary to differentiate this anomaly of the heart, which I have recently been the first to study clinically, not only from other malformations, but particularly from acquired disease of the heart Its presence is revealed only by auscultation, through a physical sign with definite characteristics this consists of a long loud *murmur* (resulting from the passage of blood through the opening in the interventricular septum and directly into the pulmonary artery or the aorta, the location of which is frequently abnormal in these cases) This murmur is unaccompanied by other murmurs, begins with systole and is so prolonged that it entirely occupies the period of the natural tic-tac of the normal heart sounds Its point of maximal intensity is not at the apex (as in the case of lesions of the auriculoventricular orifices), nor at the base on the right side (as in stenosis of the aortic orifice), but over the upper third of the precordial area It is mainly medial in location like the septum itself, and from this focal point diminishes uniformly in intensity as the stethoscope is moved over the chest The murmur is not propagated into the vessels It coincides with no other sign of organic disease with the exception of the *harsh thrill* which accompanies it This murmur is *the pathognomonic sign of a defect in the interventricular septum*

3 The differential diagnosis of this malformation (up to the present time either unrecognized or confused with other congenital or acquired lesions) will from now on be rendered simple by careful comparison of the physical signs These signs vary in number, location, and character in heart disease when structural alterations are multiple, progressive, and changing, while the murmur under discussion, like the permanently fixed lesion responsible for its occurrence, remains unaltered for an indefinite period of time The same statement holds true when comparing this murmur with the signs of functional disturbances, such signs vary with the changing episodes of heart weakness, and are entirely different in their acute or chronic charac-

teristics from the unaltered signs of a defective interventricular septum which change inappreciably over the years and increase only very gradually and almost without detection

4 The consideration of the age of the patient is a noteworthy point in the diagnosis, endocarditis, for example, is rarely seen in infancy, before the age of two years, and furthermore, the anemia of very young children is very seldom associated with a heart murmur Thus, a *murmur in a nursing infant* is almost always a definite indication of an *anomaly* of the heart or great vessels

5. The *prognosis* is generally less significant in the abnormality described than in other structural diseases of the heart, in which the danger for children is greater and occurs sooner, permitting hope for not much more than another decade of life In spite of the existence of an uncomplicated defect of the interventricular septum, patients may attain or even surpass the average span of human life

6 A definite diagnosis usually demands an active sustained program of *treatment* in heart disease But, if a congenital anomaly of the heart exists, vigorous treatment is of no avail and may even be harmful To show, thanks to the accuracy of diagnosis, when to act in one and when to withhold treatment in another, is to be of service not only to physicians but also to patients

COMMUNICATION CONCERNING CONGENITAL PATENCY OF THE INTERVENTRICULAR SEPTUM*

By

HENRI ROGER

In the paper which I read to the Academy last October 21, I sought to establish the distinct clinical existence of a cardiac anomaly not recognized up to the present, and confused, sometimes, with other congenital anomalies, sometimes with heart disease properly speaking, namely, *congenital communication of the two hearts by patency of the interventricular septum*. I presented a differential diagnosis which arises almost exclusively from auscultation and I stated that a *special cardiac murmur* was the *pathognomonic sign of this malformation*.

Two objections have been made: the first upon the *anatomical site of the murmur* which I attributed to the passage of blood across the perforation of the septum in its superior portion, that it might also as well (they tell me) be produced by the passage of blood across the foramen ovale or ductus arteriosus. For the other, it has been objected that my clinical description was not based upon a sufficient number of necropsies.

For completing my answer to these objections, I request permission of the Academy to read, in summary, an *autopsy report* which Dr Gaston Decaisne published in *Progrès Médical*, under the title, "Congenital communication of the ventricles of the heart," presented to the Anatomic Society in July 1877.

"In a little girl of 26 months (D—) who entered the Hôpital des Enfants on the service of M. Bouchut, who did not appear to have any heart disease and who was *not at all cyanotic*, auscultation revealed *an intense systolic murmur* occupying the entire precordial region, it was heard on the right side of the chest and even in the posterior thoracic region. The maximum of this murmur was over the *base and middle portion of the heart*. There was considerable bulging with a *purring thrill*. The pulse was 102 per minute, moreover it was regular, not intermittent. Cardiac dulness was not appreciably enlarged.

"In the absence of general symptoms the diagnosis of acute cardiac affection could be discarded and the existence of a *congenital lesion* was admitted. But where was the lesion? The *absence of cyanosis* excluded

*Bulletin de l'Académie de Medecine, 2me série, Tome VIII, 1879, pp 1189-1191

stenosis of the pulmonary artery There was nothing to choose from except *coarctation of the aorta* and *patent foramen ovale* Finally the last diagnosis was accepted, with reservations however

"The child died about six weeks later, after some pulmonary mishap

"*Autopsy*—The *heart* is not hypertrophied The *aorta* and *pulmonary artery,* by no means narrowed, are perhaps slightly *dilated,* but their valves are normal The mitral and tricuspid valves are likewise The ventricular walls show no alteration but *in the upper portion of the interventricular septum,* beneath the mitral valve, is found an *orifice* which establishes a *communication between the two ventricles* On the side of the left ventricle the opening is sinuous, on the side of the right ventricle it is on the contrary curved and prominent The endocardium at this level is whitish, thickened and opaque The *foramen ovale is obliterated* "

In this important observation which is interesting and rare and which does not lack the control of pathologic anatomy, was found the congenital lesion of the interventricular septum which I have separated from other malformations, the particular murmur which characterises it, and the coincidence of positive and negative signs of which I have shown the clinical picture This observation gives me proof on all points, I wish to thank my young and distinguished colleague who was so kind as to send it to me

1879

WILLIAM MURRELL

INTRODUCTION OF NITROGLYCERIN IN THE TREATMENT OF ANGINA PECTORIS

WILLIAM MURRELL

(1853-1912)

WILLIAM MURRELL was born in London on November 26, 1853, the son of William Kenrick Murrell, a lawyer He received his primary education at Murray's School in Wimbledon and his academic training at University College, London, where he was awarded the William Sharpey Physiological Scholarship and where he became a demonstrator in physiology In 1874 he qualified for the licentiate of the Society of Apothecaries, and in 1875 he qualified for membership in the Royal College of Surgeons, England, and obtained the licentiate of the Royal College of Physicians of London In 1877 he passed the qualifications for membership in the Royal College of Physicians That year, also, he was appointed medical registrar to the Westminster Hospital, having served previously as a teacher of histology He studied medicine at the University of Brussels and in 1879 received from that institution the degree of Doctor of Medicine He was elected a fellow of the Royal College of Physicians in 1883

Murrell devoted much of his lifetime to the study of pharmacology and therapeutics Early in his career he came under the influence of the British physiologist, Sydney Ringer (1835-1910), who at that time was making extensive experimental researches on the action of various drugs on living protoplasm At this period the leading physicians who served on the staffs of hospitals did not bother with the details of the prescribing of drugs, but left the task to house physicians They also showed no concern about either the physiologic or the pharmaceutic action of drugs Hence, they did not bother to encourage elementary instruction in these subjects or to avail themselves of chances to accomplish the fundamental research which was badly needed

Murrell often expressed disappointment about such a state of affairs, and even though his colleagues discouraged him, he continued his pharmacologic studies

In 1879 appeared his first independent publication, "Nitroglycerine as a Remedy for Angina Pectoris" This important and original observation we are including in our classics When this paper was published, Murrell was lecturer on practical physiology at the Westminster Hospital and was assistant physician to the Royal Hospital for Diseases of the Chest In 1883, he was appointed assistant physician, and fifteen years later became physician, to Westminster Hospital

Murrell's best known work is his "Manual of Pharmacology and Therapeutics," first published in 1896 His interest in toxicology was paramount at a time when the medical profession showed little interest in the subject Nevertheless, his book, "What to do in the Case of Poisoning," went through eleven editions before his death

Murrell was also instrumental in exposing the scandalous massage establishments which at one time flourished in the West End of London He tried, without too much success, to place massage on a scientific basis and in 1886 published his work, "Massotherapeutics, or Massage as a Mode of Treatment" However, it was many years before the profession realized the importance of his early efforts in this field

In later years, Murrell took an active interest in bacteriotherapeutics

At the time of his death, he was a member of the Faculty of Medicine of the University of London In 1881 he was elected a laureate of the Academy of Medicine of Paris In 1887 he served as one of the vice-presidents of the International Medical Congress, which was held that year in Washington, D C For some years, he served on the medical staff at the Paddington Green Children's Hospital, where he later became senior physician

For several months previous to his death, Murrell had suffered from heart disease In the spring of 1912, dropsy supervened and he was obliged to go away for a rest As soon as he felt better he returned to his medical practice Soon he had a second breakdown He suffered from extreme hypertrophy and dilatation of the heart with complications, which resulted in his death on June 28, 1912, at the age of fifty-eight

NITRO-GLYCERINE AS A REMEDY FOR ANGINA PECTORIS*

By

WILLIAM MURRELL, M R.C P.

Lecturer on Practical Physiology at Westminster Hospital, and Assistant-Physician to the Royal Hospital for Diseases of the Chest

SOME TWENTY years ago a controversy took place in the pages of the *Medical Times and Gazette*, on the properties, physiological and therapeutical, of the substance known to chemists as nitro-glycerine The discussion was opened by Mr A G Field, then of Brighton, who described in detail the symptoms he had experienced from taking two drops of one per cent solution of nitro-glycerine in alcohol About three minutes after the dose had been placed on his tongue, he noticed a sensation of fulness in both sides of the neck, succeeded by nausea For a moment or two there was a little mental confusion, accompanied by a loud rushing noise in the ears, like steam passing out of a tea-kettle He experienced a feeling of constriction around the lower part of the neck, his forehead was wet with perspiration, and he yawned frequently These sensations were succeeded by a slight headache and a dull heavy pain in the stomach, with a decided feeling of sickness, though without any apprehension that it would amount to vomiting He felt languid and disinclined for exertion, either mental or physical This condition lasted for half an hour, with the exception of the headache, which continued till the next morning These symptoms Mr Field described as resulting from a single dose of one-fiftieth of a grain Thinking that possibly he might be unusually susceptible to the action of the drug, he induced a friend to take a dose The gentleman experienced such decided effects from merely touching his tongue with the cork of the bottle containing the nitro-glycerine solution that he refused to have anything more to do with it A lady suffering from toothache, on whose tongue Mr Field placed about half a drop of the same solution, experienced a pulsation in the neck, fulness in the head, throbbing in the temples, and slight nausea The toothache subsided and she became partly insensible, disliking much to be aroused When fully sensible she had a headache, but the toothache was gone Another of Mr Field's patients, a stout, healthy young woman, accidentally swallowed a small piece of lint dipped in the nitro-glycerine, whilst being applied to a decayed tooth In about five minutes,

*Lancet 1 80-81 113-115 151-152 225-227 1879

after feeling giddy and sick, with headache, she became insensible. Her countenance, naturally florid, was unaltered, breathing tranquil, pulse full, and rather quickened. She recovered in about three minutes, after the administration of a stimulant. Some headache was complained of, but the toothache was gone. Mr. Field, in conclusion, offered some suggestions as to the therapeutical uses of the drug, and stated that he had not met with a single well-defined case of neuralgia or spasmodic disease in which it had failed to afford some relief.

This paper was followed by a letter from Dr. Thorowgood, in the main confirmatory of Mr. Field's observations. He, after taking a small dose, experienced "a tensive headache over the eyes and nose, extending also behind the ears, and soon followed by a tight, choking feeling about the throat, like strangulation. Neither loss of consciousness nor nausea was experienced, and a walk by the sea soon did away with the unpleasant feeling."

These statements did not long remain unchallenged, their accuracy being called in question by Dr. George Harley, of University College, and Dr. Fuller, of St. George's. Dr. Harley, having obtained some nitro-glycerine of the same strength as Mr. Field's, commenced his observations by touching his tongue with the cork of the bottle containing the solution. He experienced "a kind of sweet and burning sensation, and soon after a sense of fulness in the head, and slight tightness about the throat, without, however, any nausea or faintness." After waiting a minute or two these effects went off, and Dr. Harley was inclined to think "they were partially due to imagination." Determined, however, as he says, to give the drug a fair chance, he swallowed five drops more, and as this did not cause any increased uneasiness, he took, in the course of a few minutes, another ten drops of the solution. Being at the time alone he became alarmed lest he should have taken an over-dose, and very soon his pulse rose to above 100 in a minute. The fulness in the head and constriction in the throat were, he thought, more marked than after the smaller dose. In a minute or two the pulse fell to 90, but the fulness in the head lasted some time, and was followed by a slight headache. To two medical friends Dr. Harley administered respectively twenty-eight and thirty-eight drops in divided doses without the production of any symptoms. Some pure nitro-glycerine was then obtained, and of this Dr. Harley took, in the course of a few minutes, a drop, equivalent to a hundred drops of the solution previously employed. The only symptoms produced were a quickened pulse, fulness in the head, and some tightness in the throat, but as these passed off in a few minutes, Dr. Harley considered that they were probably the effects of "fear and imagination." On a subsequent occasion he took, in the course of three-quarters of an hour, a quantity of the nitro-glycerine solution equivalent to 199½ drops of the solution used by Mr. Field, with the production of no more dis-

agreeable symptoms than those he had experienced in his former trials The quickening of the heart's action he ascribed to fear, but the head and neck sensations were, he considered, "too constant to be attributed to the same cause," although he thought they were exaggerated by the imagination Dr Harley, in conclusion, states that he experimented on ten different gentlemen with nitro-glycerine solution, obtained from four different sources, without witnessing any dangerous effects when administered in the above doses, but he adds that, if taken pure, great caution should be used

In a second communication to the same journal Mr Field reasserted the correctness of his observations, and maintained that a reasonable explanation of the very different results obtained by different observers might be found in the great variation in strength to which the drug is liable He considered, too, that the conditions under which the drug was taken had much to do with its action When the system is worn out by fatigue, he says, it is more likely to act powerfully than when taken under less unfavourable conditions On the occasion of taking the dose which produced in him such startling effects, his nervous energy had been impaired by an unusually hard day's work He found that under more favourable conditions he could take the same dose with production of nothing worse than headache Having in his experiments on himself experienced the greatest variation in the strength of different specimens of nitro-glycerine, he was disposed to think, on reading the account given by Dr Fuller and Dr Harley, that they had used a less powerful agent He accordingly called on Dr Fuller, and induced him to take a dose of the solution he had used, but, to his surprise, he experienced little beyond headache On the same day Mr Field administered to a hospital patient suffering from hemicrania two drops of the solution In about a minute he became pallid, felt sick and giddy, his forehead was covered with perspiration, and he sank on the bed by which he was standing almost unconscious, his pulse failing so as scarcely to be felt After the administration of a little ammonia the circulation became more vigorous, and in twenty minutes there was a marked diminution of the pain, and he experienced a great desire to sleep, a luxury of which his sufferings had almost deprived him on previous nights Mr Field administered small doses of the drug to several other people, all of whom were distinctly affected by it

Being greatly interested in this curious controversy, and being quite at a loss to reconcile the conflicting statements of the different observers, or arrive at any conclusion respecting the properties of the drug, I determined to try its action on myself Accordingly I obtained some one per cent solution One afternoon, whilst seeing out-patients, I remem-

bered that I had the bottle in my pocket. Wishing to taste it, I applied the moistened cork to my tongue, and a moment after, a patient coming in, I had forgotten all about it. Not for long, however, for I had not asked my patient half a dozen questions before I experienced a violent pulsation in my head, and Mr. Field's observations rose considerably in my estimation. The pulsation rapidly increased, and soon became so severe that each beat of the heart seemed to shake my whole body. I regretted that I had not taken a more opportune moment of trying my experiments, and was afraid the patient would notice my distress, and think that I was either ill or intoxicated. I was quite unable to continue any questions, and it was as much as I could do to tell him to go behind the screen and undress so that his chest might be examined. Being temporarily free from observation, I took my pulse and found that it was much fuller than natural and considerably over 100. The pulsation was tremendous and I could feel the beating to the very tips of my fingers. The pen I was holding was violently jerked with every beat of the heart. There was a most distressing sensation of fulness all over the body and I felt as if I had been running violently. I remained quite quiet for four or five minutes and the most distressing symptoms gradually subsided. I then rose to examine the patient, but the exertion of walking across the room intensified the pulsation. I hardly felt steady enough to perform percussion and determined to confine my attention to auscultation. The act of bending down to listen caused such an intense beating in my head that it was almost unbearable and each beat of the heart seemed to me to shake not only my head, but the patient's body too. On resuming my seat I felt better and was soon able to go on with my work, though a splitting headache remained for the whole afternoon. Were my symptoms due to nervousness or anxiety? Certainly not. I will not say that I discredited Mr. Field's observations, but after Dr. Harley's positive assertions I certainly did not expect to obtain any very definite results from so small a dose. Moreover, at the moment of the onset of the symptoms I was engaged in the consideration of another subject and had forgotten all about the nitro-glycerine. I did nothing to intensify the symptoms, but, on the contrary, should have been only too glad to have got rid of them. The headache, I can most positively affirm, was anything but fancy. Since then I have taken the drug some thirty or forty times, but I never care to do so unless I am quite sure that I can sit down and remain quiet for a time, if necessary. It uniformly produces in me the same symptoms, but they are comparatively slight if I refrain from moving about or exertion of any kind. The acceleration of the pulse is very constant, although sometimes it amounts to not more than ten beats in the minute. The temperature remains unaffected. The pulsation is often so severe as to be acutely painful. It jerks the whole body so that a book held in the hand is seen to move quite distinctly at each beat of the heart.

The amount of pulsation may be roughly measured by holding a looking-glass in the hand and throwing the reflection into a dark corner of the room. Before taking the drug the bright spot may be kept steady, but as soon as the pulsation begins it is jerked violently from side to side. I have taken all doses from one minim to ten, sometimes simply dropped on the tongue, at others swallowed on sugar or in water. I have not ventured to take more than fifteen minims in a quarter of an hour. Once or twice a ten minim dose has produced less pulsation than I have experienced at other times in a single drop, but then with the larger quantity one is careful to avoid even the slightest movement. After a five minim dose I usually experience a certain amount of drowsiness—a lazy contented feeling, with a strong disinclination to do anything.

Thinking there might be individual differences of susceptibility to the action of nitro-glycerine, I have laid my friends and others under contribution and have induced as many as possible to give it a trial. I have notes of thirty-five people to whom I have administered it, twelve males and twenty-three females, their ages varying from twelve to fifty-eight. I find they suffered from much the same symptoms as I did, although it affects some people much more than others. Of the numbers above quoted, only nine took minim doses without experiencing decided symptoms. Women and those below par are much more susceptible to its action than are the strong and robust. A delicate young lady, to whom, adopting Mr. Field's suggestion, I administered it in drop doses for the relief of neuralgia, experienced very decided effects from it, each dose producing a violent headache lasting from half an hour to three hours. A married woman, aged thirty-five, took one minim with very little inconvenience, but was powerfully affected by two. She was obliged to sit down after each dose and was positively afraid to move. It made her hot and caused such a beating in her head that she had to support it with her hands. She experienced a heavy weight on the top of the head and also a sharp darting pain across the forehead, which for a moment or two was very painful to bear. A friend, who for some days took four drops every three or four hours, informs me that at times it affected his head "most strangely." The pulsation was very distressing and often lasted an hour or more, being intensified by moving. It has relieved him of an old-standing facial neuralgia, and he is enthusiastic in its praise. A young woman, aged twenty-nine, complained that after every dose of the medicine—one minim—"it seemed as if the top of her head were being lifted off," and this continued sometimes for five minutes and sometimes longer. The medicine made her bewildered, and she felt sick. A patient with a faint apex systolic murmur was ordered one minim in half an ounce of water four times a day. He took two doses, but it caused "such a beating, thumping, hot pain" in his head that he was unable to continue it. A young man who was given nitro-glycerine in mistake for

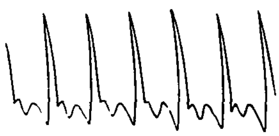
phosphorus said it made his temples throb, and he could see his pulse beat so distinctly that he was frightened It caused a burning and flushing in his face, and "took every bit of strength away" This would last for twenty minutes or half an hour after each dose There was no headache That alarming symptoms may be produced by large doses, is shown by the following case A woman, aged fifty-one, was ordered drop doses of the one per cent solution every four hours This was taken well, and at the expiration of a week, the dose was doubled No complaint being made, it was then increased to four minims, and after a time to six The patient said "the medicine agreed with her," and even leading questions failed to elicit any complaint of headache or the like After the medicine had been taken continuously for five weeks the dose was increased to ten minims The patient then stated that the medicine no longer agreed with her, it made her sick after every dose and took her appetite away She always vomited about five minutes after taking the medicine, the vomiting being immediately followed by headache The medicine made her "go off in a faint" after each dose She had three "fainting fits" in one day and could not venture to take another dose She became quite insensible and once remained so for ten minutes Each fainting fit was "followed by cold shivers," which "shook her violently all over" Her husband and friends were greatly alarmed, but she thought on the whole it had done her good She had never noticed that the medicine produced drowsiness In another case a three minim dose taken on an empty stomach caused a feeling of faintness, "everything goes dark," the patient said, "just as if I were going to faint" The patient could take the same dose after meals without the production of any unpleasant symptoms Drowsiness is not an uncommon result of taking nitro-glycerine

From a consideration of the physiological action of the drug and more especially from the similarity existing between its general action and that of nitrite of amyl, I concluded that it would probably prove of service in the treatment of angina pectoris, and I am happy to say that this anticipation has been realised

As a preliminary step I was anxious to obtain a comparative series of sphygmographic tracings Judged by the sphygmographic tracings, the effects of nitrite of amyl and of nitro-glycerine on the pulse are similar Both drugs produce a marked state of dicrotism and both accelerate the rapidity of the heart's action They differ, however, in the time they respectively take to produce these effects The full action of the nitro-glycerine is not observed in the sphygmographic tracings until six or seven minutes after the dose has been taken In the case of nitrite of amyl, the effect is obtained in from fifteen to twenty seconds after an inhalation or a dose has been taken on sugar The influence of the nitrite of amyl is extremely transitory, a tracing taken a minute and a half after

Influence of Nitrite of Amyl on the Pulse

No. 1.—Before inhalation



No. 2.—One minute after inhalation

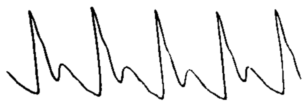
No. 3.—Two minutes after inhalation

Influence of Nitro-Glycerine on the Pulse

No. 1.—Before dose

No. 2.—Two minutes after dose

No. 3.—Eight minutes after dose



No. 4.—Nine minutes after dose

No. 5.—Ten minutes after dose

No. 6.—Twenty-two minutes after dose

No. 7.—Twenty-six minutes after dose

the exhibition of the drug being perfectly normal. In fact, the full effect of the nitrite of amyl on the pulse is not maintained for more than fifteen seconds. The nitro-glycerine produces its effects much more slowly, they last longer and disappear gradually, the tracing not resuming its normal condition for nearly half an hour. The effect may be maintained for a much longer time by repeating the dose. Nitro-glycerine is more lasting in its power of producing a dicrotic form of pulse beat, and consequently in cases where the conditions of relaxation and dicrotism are desired to be maintained for a space of time, its exhibition is to be preferred to that of nitrite of amyl.

During the last nine months I have treated three cases of undoubted angina pectoris with nitro-glycerine with what success the cases themselves will show.

William A—, aged sixty-four, first came under observation in December, 1877, complaining of intense pain in the chest, excited by the slightest exertion. It was distinctly paroxysmal, the patient being perfectly well in the intervals. The first attack was experienced in September, 1876. Patient was at the time in his usual health and was, in fact, out for a day's pleasure in the country. The pain seized him quite suddenly when walking. It was a most severe attack—as severe a one as ever he experienced in his life. It caused both him and his friends great alarm and they were most anxious that he should return home at once. He cannot tell at all what brought it on, he had been enjoying himself very quietly, it was not by any means a cold day, and he had not been running, or even walking faster than usual. He remained perfectly well until the following April when he experienced another similar attack and since then he has been suffering from them with increasing frequency. From September, 1877, they have been a source of constant anxiety and it was only by a determined effort that he could continue to follow his occupation.

The attacks usually commence with a feeling of warmth, then of heat, and then of burning heat in the chest immediately followed by a heavy pressure, from the midst of which proceeds an acute pain, so that in a moment the whole chest seems as if it were one mass of pain. It is almost impossible, he says, to describe it for he never felt anything like it before. The pain is first experienced at a small spot on either side of the sternum, corresponding to its junction with the fourth costal cartilages. From the chest the pain flies to the inner side of the arm at a point midway between the shoulder and the elbow. It runs down as far as the elbow, but never to the fingers. It is not more severe on one side than the other. During the seizure the patient suffers most acutely and feels convinced that some day he will die in an attack. He usually experiences some shortness of breath at the time, but there is no feeling of

constriction about the chest He can speak during the seizure, though with some difficulty The attacks are not accompanied by any sensation of warmth or chilliness, but patient is under the impression that he grows pale at the time These attacks are induced only by exertion in some form or other, most commonly by walking, and especially by walking fast Walking up hill is sure to bring on a seizure Stooping down has a similar effect and the act of pulling on the boots will excite a paroxysm almost to a certainty He is almost afraid to stoop down and when he wants to pick up anything from the floor he goes down on his hands and knees He has a slight cough, but although it shakes him at times, it never brings on the pain The attacks are not excited by food, but exercise taken after meals is more likely to induce them than when taken on an empty stomach Patient has noticed that they are far more readily excited immediately after breakfast than at any other period of the day

There could be no possibility of doubt respecting the diagnosis It was a typical uncomplicated case of angina pectoris

Patient was placed for a week on infusion of quassia in order that he might be observed and also to eliminate the effects of expectation It need hardly be said that he derived no benefit from this treatment He was then ordered drop doses of the one per cent nitro-glycerine solution in half an ounce of water three times a day At the expiration of a week he reported that there had been a very great improvement The attacks had been considerably reduced in frequency and for two or three days he had had only one attack in the morning after breakfast The attacks, when they did occur, were much less severe He found, too, that a dose of medicine taken during an attack would cut it short He had tried it several times, and it had always succeeded It would not act instantly, but still very quickly, so that the attacks were considerably shortened He was thoroughly convinced that the medicine had done him good and said he was better than he had been since first he had the attacks

Patient had adopted the plan of carrying his medicine with him in a phial and taking a dose if an attack seized him in the street It never failed to afford relief

1885

PIERRE CARL ÉDOUARD POTAIN

DESCRIPTION OF THE MECHANISM OF GALLOP RHYTHM

THE THEORY OF GALLOP RHYTHM*

By M. POTAIN

All the theories which attempted to explain the mechanism of gallop rhythm were unacceptable for clinical considerations. M. Potain undertook to establish the following theory, which he supported by graphic demonstrations.

The gallop stroke is diastolic and is due to the beginning of sudden tension in the ventricular wall as a result of the blood flow into the cavity.

It is more pronounced if the wall is not distensible and the failure of distensibility may depend either on a sclerotic thickening of the heart wall (hypertrophy due to Bright's disease) or to decrease in muscular tonicity. Since the wall, by virtue of its own elasticity, is no longer able to resist the inflow of blood, it is placed under tension precisely at the same moment that this occurs.

The gallop can originate in all cases where the elastic resistance of the wall encroaches on muscular tonicity, either by an increase of the first factor, or a diminution of the second.

It was observed in a goodly number of acute diseases, chiefly in typhoid fever, and also in cachectic subjects, in whom cardiac function is embarrassed. It occurs either in a constant or more pronounced manner in ventricular hypertrophy of Bright's disease, or on the other hand, accompanies dilatation of the right heart of hepato-gastro-intestinal origin. It is therefore an important sign owing to the latent or insidious character of the latter diseases.

The name, gallop rhythm, was first introduced by Bouillaud† and should be used for the phenomenon to which it applies, the phenomenon does not, however, always maintain the character of the gallop of a horse.

One could apply the name of murmur of diastolic shock in all the cases in which the above mentioned theory is applicable and reserve the term, gallop rhythm, specifically for the type, in which the *anapest* rhythm occurs.

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*Comments in Assoc. française pour l'Avancement des Sciences. Compt. rend., pt. 1, 14: 201-203, 1885.

For an account of Potain's life see page 531. Potain's paper "Description of the Pulsations in the Jugular Veins" is reprinted on pp. 533-556.

†See page 446.—F. A. W. 1940.

1887

AUGUSTUS D. WALLER

DEMONSTRATION OF A METHOD OF LEADING OFF THE ACTION CURRENTS OF THE HEART BY MEANS OF CONTACT ELECTRODES

AUGUSTUS DÉSIRE WALLER

(1856 1922)

AUGUSTUS DÉSIRE WALLER was born in Paris on July 12, 1856 He was the only child of Dr Augustus Volney Waller, a physiologist of international reputation Dr Waller, Senior, had observed the emigration of leucocytes in 1846, and had described the ciliospinal region in 1851 In 1853 he had emphasized the vasoconstrictor action of the sympathetic nerves, and in 1856 he had observed that nerves, when separated from their cells of origin, undergo degeneration

Young Waller received his academic training at the Collège de Genève On the death of his father in 1870, the widow and son moved to Aberdeen, Scotland In due time Waller matriculated at the University of Aberdeen, where he began the study of medicine At Aberdeen in 1878 he received the medical degrees, Bachelor of Medicine and Master of Surgery He continued his medical training at the University of Edinburgh, where, according to the editor of the ''Lancet,'' he received the degree of Doctor of Medicine in 1881

Soon after graduation, Waller went to London, where he worked under Burdon-Sanderson in his physiologic laboratory In 1880 and again in 1883 he received scientific grants from the British Medical Association In 1884, he was appointed a research scholar About this time he received the appointment of lecturer on physiology at the London School of Medicine for Women There he met, and afterwards married, Alice Mary, the second daughter of George Palmer of Reading, a member of Parliament

Soon after his marriage, Waller was appointed lecturer on physiology in the Medical School of St Mary's Hospital, where he taught for sixteen years In 1902, the Senate of the University of London established a physiological laboratory and Waller was appointed its director There he continued a brilliant career until his death in 1922 He died at his home in St John's Wood on March 11, 1922, as the result of a cerebral hemorrhage

Waller's most important contributions to medicine were in the field of electro physiology He was the first to demonstrate that the currents set up by the beating of the heart in animals could be recorded without opening the thorax He was the first, also, to obtain an electrocardiogram of the action of the human heart We are reprinting his first classic contribution in this field, a demonstration of leading off the action currents of the heart by means of contact electrodes This paper appeared in the ''Journal of Physiology'' for 1887

A summary of Waller's electrophysiologic work may be found in his Oliver Sharpey Lectures on the electrical action of the human heart, delivered before the Royal College of Physicians in 1913

In the early part of his career, Waller was much interested in investigating the electric currents in living structures, especially in nerve and muscle, but also in the skin, the retina, and in plants In 1903 he summarized these observations in his book, ''Signs of Life from their Electrical Aspect ''

Waller is also remembered for his observations on the effects of gases and anesthetic vapors on the irritability of nerves and muscles Having been a fellow of

the Royal Society since 1892, he was asked, in 1896, to deliver the Croonian Lecture before that society In this lecture he expressed his ideas concerning his studies of isolated nerves In 1897, as president of the Section on Anatomy and Physiology of the British Medical Association, he read a paper on the relative efficacy of ether and chloroform as anesthetics

During his last years, Waller was primarily interested in two subjects of importance, the physiologic cost of muscular work as measured by the amount of carbon dioxide exhaled, and the emotive response of man to pain or the threat of pain

Waller was the author of an important textbook, the ''Introduction to Human Physiology,'' first published in 1891 and later in several other editions He also contributed several articles to the ''Transactions'' of the Royal Society and to the ''Journal of Physiology'' In 1909 he delivered the Hitchcock Lectures at the University of California

For his outstanding scientific contributions Waller received, besides many national honors, membership in many scientific societies abroad He was a corresponding member of the Société de Biologie, Paris, a member of the Physiological Society of Moscow, and of the Royal Academies of Medicine of Rome and Brussels For his electrophysiologic work he was elected a lauréat de l'Institut de France

A DEMONSTRATION ON MAN OF ELECTROMOTIVE CHANGES ACCOMPANYING THE HEART'S BEAT*

By

AUGUSTUS D WALLER

IF A PAIR of electrodes (zinc covered by chamois leather and moistened with brine) are strapped to the front and back of the chest and connected with a Lippmann's capillary electrometer, the mercury in the latter will be seen to move slightly but sharply at each beat of the heart[1] If the movements of the column of mercury are photographed on a travelling plate simultaneously with those of an ordinary cardiographic lever, a record is obtained as under Fig 1, in which the upper line *h h* indicates the heart's movements and the lower line *e e* the level of the mercury in the capillary Each beat of the heart is seen to be accompanied by an electrical variation

The first and chief point to determine is whether or no the electrical variation is physiological and not due to a mechanical alteration of contact between the electrodes and the chest wall caused by the heart's impulse To ascertain this point, accurate time-measurements are necessary, a physiological variation should precede the movement of the heart, while this could not be the case if the variation were due to altered contact Fig 2 is an instance of such time-measurements taken at as high a speed of the travelling surface as may be used without rendering the initial points of the curves too indeterminate It shows that the electrical phenomenon begins a little before the cardiographic lever begins to rise The difference of time is, however, very small, only about 0 025″, and this amount must further be diminished by 0 01″ which represents the "lost time" of the cardiograph The actual difference is thus no greater than 0 015″, and the record is therefore, although favourable to the physiological interpretation, not conclusively satisfactory

We know from the experiment of the secondary contraction made by Helmholtz[2] on voluntary muscle, by Kolliker and Muller[3] and by Donders[4] on the heart, that the negative variation of muscle begins before

*J Physiol 8 229-234 1887

[1]The capillary column of mercury is vertical the electrode connected with the mercury will be spoken of as the Hg electrode that connected with the sulphuric acid as the H_2SO_4 electrode When the Hg electrode becomes negative to the H_2SO_4 electrode the mercury moves away from the point of the capillary—i e northwards in the field of the microscope with eye-piece or when projected without eye-piece When the H_2SO_4 electrode becomes negative to the Hg electrode the movement of the mercury is southwards The letters S and N will be used to characterise such movements When these movements are photographed the mercury interrupting the light of a vertical slit gives the white portion of the developed sensitive plate the remaining portion being black with the exception of the white lines caused by the chronographic or other levers which throw their shadows across the upper part of the slit The tracings read from left to right

[2]Monatsberichte Berlin Acad 1854 p 329

[3]Verhandl phys und Ges Wurtzburg 1856 VI p 528

[4]Onderzoekingen Utrecht 1872 p 261

its visible movement, and the current of action of the heart begins before the commencement of the heart's contraction For muscle, the time-difference given is 1/200″, for the heart (rabbit) 1/70″, for the frog's heart, the rheotome observations of Marchand[1] are to the effect that the variation begins 0 01″ to 0 04″ after excitation, while the contraction does not begin until 0 11″ to 0 33″ The capillary electrometer may with advantage be employed to measure this time-difference, the electrical and the mechanical events being simultaneously recorded This I carried

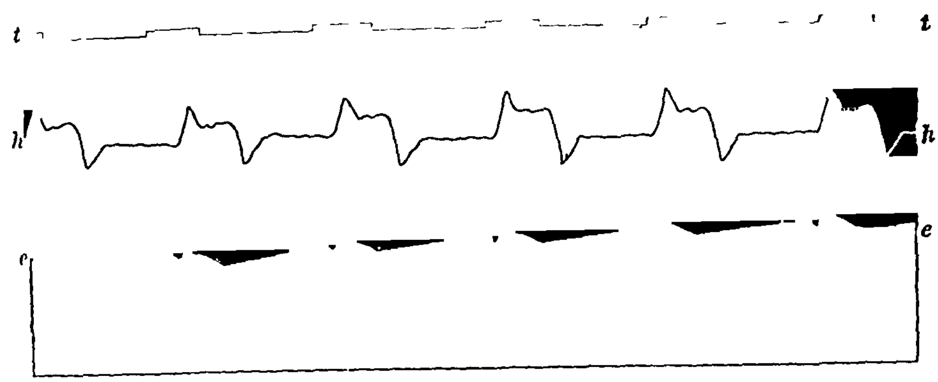

FIG 1 Man Heart led off to electrometer from front and back of chest (front to Hg, back to H_2SO_4)
e e electrometer *h h* cardiograph *t t* time in seconds

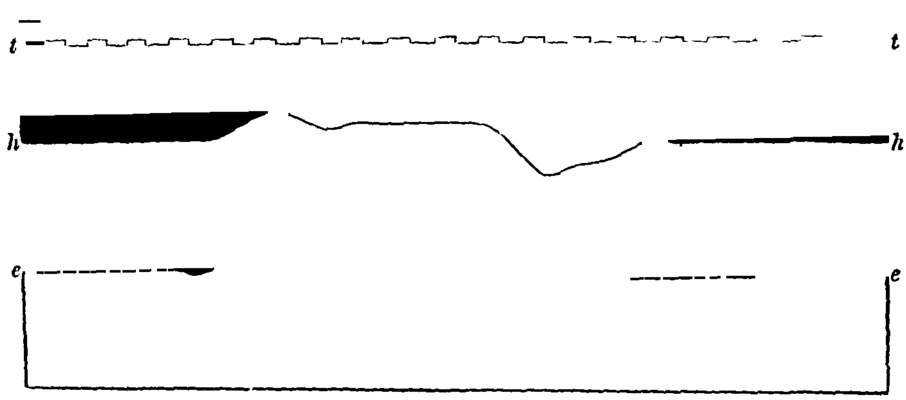

FIG 2 Man Heart led off to electrometer from front and back of chest (front to Hg, back to H_2SO_4)
e e electrometer *h h* cardiograph *t t* time in 1/20th sec

out on voluntary and upon cardiac muscle with the same instrument as that which I employed for the human heart, and thus ascertained that its indications are trustworthy in this capacity

In all these cases the antecedence of the electrical variation is clear and measurable In the case of the excised kitten's heart, the time-difference is about 0 05″ with a length of contraction of about 2″, i e, the interval between the electrical and the mechanical event is increased in the sluggishly acting organ In the case of the human heart, the time-difference appears to be about 0 015″ with a length of systole of 0 35″—

[1] Pflüger's Archiv XV 1877, p 511

a value which corresponds with that obtained by Donders for the rabbit's heart in situ by the method of the secondary contraction, viz $1/70''$ (the length of systole being presumably about $1/3''$)

That a true electrical variation of the human heart is demonstrable may further be proved beyond doubt by leading off from the body otherwise than from the chest wall If the two hands or one hand and one

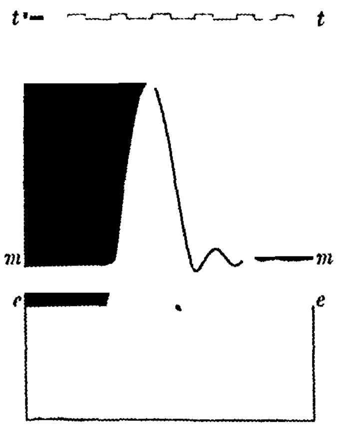

Fig 3 Frog Gastrocnemius led off to electrometer from the middle of the muscle and from the tendon Contraction excited by a single break induction shock applied to the sciatic nerve

e e electrometer *m m* muscle *t t* time in $\frac{1}{20}$th sec

(muscle to H_2SO_4, tendon to Hg)

The diphasic variation (1st phase middle negative to end, 2nd phase end negative to middle) begins about 01″ before the commencement of muscular contraction

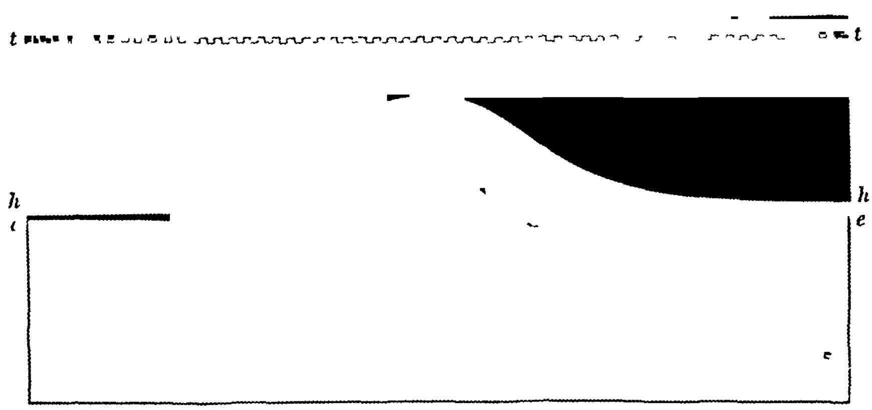

Fig 4 Frog's heart Spontaneous contraction

e e electrometer *h h* heart's contraction *t t* time in $\frac{1}{20}$th sec

(apex to H_2SO_4, base to Hg)

The variation is diphasic—S N

The first phase begins $\frac{1}{10}''$ before the commencement of contraction

foot be plunged into two dishes of salt solution connected with the two sides of the electrometer, the column of mercury will be seen to move at each beat of the heart, though less than when electrodes are strapped to the chest The hand and foot act in this case as leading off electrodes from the heart and by taking simultaneous records of these movements of

the mercury and of the movements of the heart it is seen that the former corresponds with the latter, slightly preceding them and not succeeding them as would be the case if they depended upon pulsation in the hand or foot This is unquestionable proof that the variation is physiological, for there is here of course no possibility of altered contact at the chest wall, and any mechanical alteration by arterial pulsation could only produce an effect 0 15″ to 0 20″ after the cardiac impulse A similar result is obtained if an electrode be placed in the mouth while one of the extremities serves as the other leading off electrode The electrical variation precedes the heart's beat as in the other cases mentioned

In conclusion it will be well to allude to the difficulties which arise in the interpretation of the character of the electrical variation of the human heart

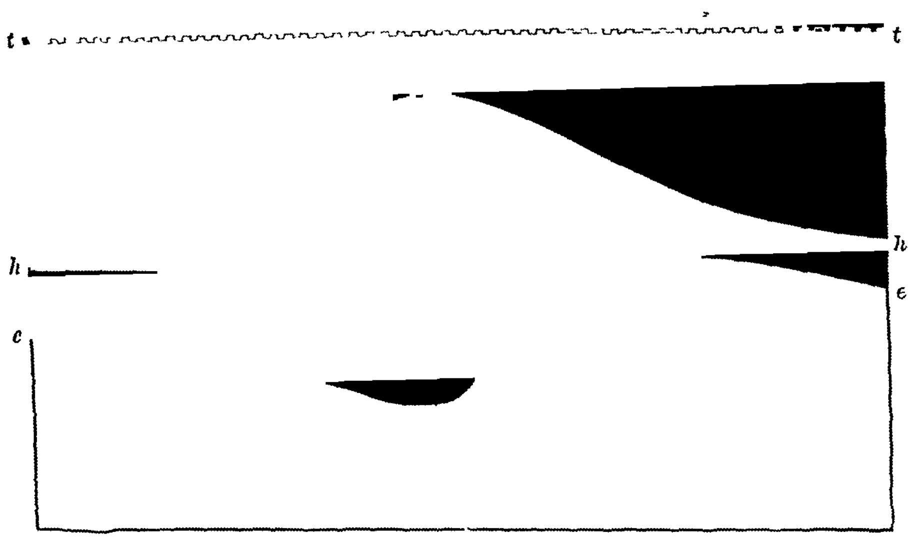

FIG 5 Kitten's heart, excised
(apex to Hg base to H_2SO_4)
e e electrometer *h h* cardiograph *t t* time in $\frac{1}{20}$th sec

By mere inspection of the electrometer, it is often most difficult to determine the direction of very rapid movements of the mercury, and photography must be employed But even then, owing to the small amplitude of the movement, it is still difficult to say whether the variation consists of two movements, and whether each movement indicates a single or a double variation in the same direction Differences in the position of the electrodes also give rise to differences of the apparent variation Thus with the following position of the electrodes (Hg electrode over the apex beat, H_2SO_4 electrode on the right side of the back) the variation as watched through the microscope appears usually nN, and changes to SN if the Hg electrode be shifted to the sternum If the Hg electrode is on the back and the H_2SO_4 electrode over the apex beat, the variation appears to be sS and to become nS when the H_2SO_4 electrode is shifted away from the apex beat The variations accompany-

ing the heart's beat observed as carefully as possible (without the aid of photography) on a healthy person with different positions of the leading off electrodes were as follows It is to be remarked that the direction of variation as observed in this series is not such as to indicate negativity of the cardiac electrode but the reverse

							Electrodes reversed
Precordium	to H_2SO_4	Back	to Hg	SS	variation		NN
"	"	Left hand	"	SS	"		NN
"	"	Right hand	"	SS	"		NN
"	"	Left foot	"	SS	"		NN
"	"	Right foot	"	SS	"		NN
Left hand	"	Right hand	"	SS*	"		NN
"	"	Left foot	"	?	"		?
"	"	Right foot	"	?	"		?
Right hand	"	Right foot	"	NN*	"		SS
"	"	Left foot	"	NN	"		SS
Right foot	"	Left foot	"	0	"		0
Mouth	"	Precordium	"	NN	"		SS
"	"	Right hand	"	0	"		SS
"	"	Left hand	"	NN*	"		SS
"	"	Right foot	"	NN	"		SS
"	"	Left foot	"	NN	"		SS

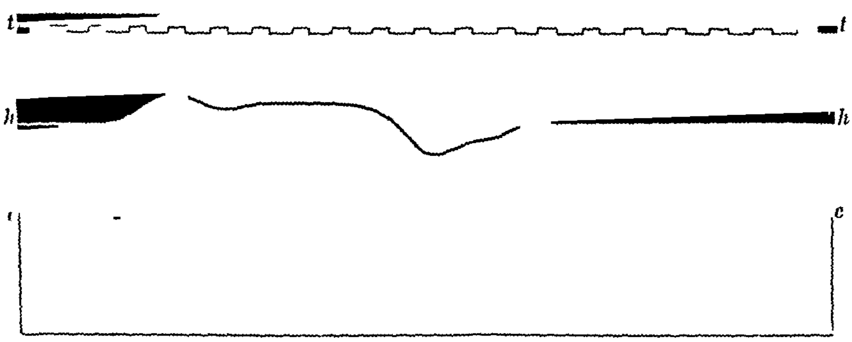

FIG 6 Man Led off to electrometer by right hand and right foot (hand to Hg foot to H_2SO_4)

e e electrometer *h h* cardiograph *t t* time in $\frac{1}{20}$th sec

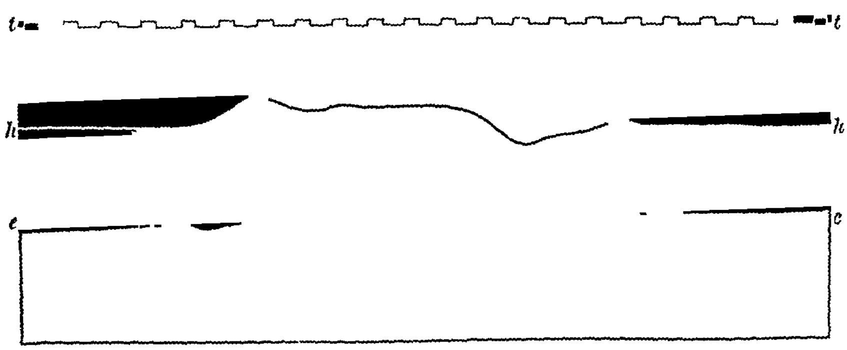

FIG 7 Man Led off to electrometer by mouth and left foot (mouth to Hg foot to H_2SO_4)

e e electrometer *h h* cardiograph *t t* time in $\frac{1}{20}$th sec

It is on account of these sources of doubt that I have not thought it advisable at this stage to attempt a definite interpretation of the character of the variation, which although as shown, especially by the experiments illustrated in Figs 6 and 7, is certainly physiological, may nevertheless be physically complicated by the conditions of demonstration on the human body

1887

JOHN A. MacWILLIAM

THE EXPERIMENTAL PRODUCTION OF EXTRASYSTOLES, VENTRICULAR FIBRILLATION AND AURICULAR FLUTTER

J. A. MacWilliam

JOHN A MacWILLIAM

(Courtesy New York Academy of Medicine)

JOHN ALEXANDER MacWILLIAM

(1857-1937)

JOHN ALEXANDER MacWILLIAM was born in 1857 in Culmill, Scotland. He studied medicine at the University of Aberdeen, where he received the degrees of Bachelor of Medicine and Master of Surgery in 1880. For his high scholastic standing he was awarded the John Murray Medal. In 1882 he received the degree of Doctor of Medicine with honors. His thesis concerned the cardiac muscular fiber in the various classes of the animal kingdom, and the diaphragmatic fiber in various animals.

MacWilliam spent some time in postgraduate work at the Universities of Edinburgh and Leipzig, and at University College in London. At the University of Leipzig it was his good fortune to work under Karl Ludwig. There MacWilliam, with Henry Pickering Bowditch (1840-1911) of Harvard, and Walter Holbrook Gaskell (1847-1914) of Cambridge, studied the physiologic properties of heart muscle.

In 1882, MacWilliam was appointed demonstrator of physiology at University College in London. He held this position until 1886, when he was appointed professor of physiology at the University of Aberdeen. In London, he had also served for some time on the staff of the Charing Cross Hospital Medical School and lectured on physiology at the London School of Medicine for Women.

When MacWilliam had been at the University of Aberdeen for not quite a year he published his important paper, ''Fibrillar Contraction of the Heart'' (1887). This paper we are reproducing for our readers. It contains his discovery that fibrillar contraction of the heart is caused by a lack of harmony in the contraction and relaxation of the minute muscular fibers which compose the walls of the heart. He showed that fibrillation is brought about by a ''rapid succession of incoordinated peristaltic contractions.'' He also described the relationship of the refractory period to this disturbance, and he gave evidence that certain poisons, when injected into the blood stream, led to the occurrence of fibrillar contractions of the ventricles. According to an editorial in the ''Lancet,'' MacWilliam, as early as 1887, believed that sudden death, during the administration of chloroform, is the result of ventricular fibrillation.

MacWilliam contributed many articles to medical literature. In 1913 he wrote an important paper for ''Heart,'' entitled, ''Blood Pressures in Man in Normal and Pathologic Conditions.'' Later, in 1925, he again contributed an article on this subject to the ''Physiological Review.''

In 1916, MacWilliam was elected a fellow of the Royal Society. Many of his papers are contained in the ''Proceedings'' of this Society, including his studies on the action of chloroform, and his studies on ether, proteins, and muscle sounds.

He retired from his professorship in 1927 to become emeritus professor, and in 1928 the University of Aberdeen conferred upon him the honorary degree of Doctor of Laws.

He died on January 13, 1937, in his eightieth year. He was married twice and is survived by his widow.

FIBRILLAR CONTRACTION OF THE HEART*

By

JOHN A. MacWILLIAM, M.D.

Professor of the Institutes of Medicine in the University of Aberdeen

MANY years ago Ludwig and Hoffa[1] showed that the application of strong constant currents or faradic currents to the ventricles of the dog's heart causes an abolition of the normal beat. The ventricular muscle is thrown into a state of irregular arhythmic contraction, whilst there is a great fall in the arterial blood pressure. The ventricles become dilated with blood as the rapid quivering movement of their walls is insufficient to expel their contents; the muscular action partakes of the nature of a rapid inco-ordinated twitching of the muscular tissue. This condition persists for a very long time in the dog, and as Ludwig showed, it is possible to kill an animal in this way—by applying a faradic current to the ventricles. The auricles go on beating rhythmically; they do not participate in the irregular movement excited in the ventricles. These phenomena are familiar to all who have worked much with the mammalian heart; they have been designated by various names—Herz-delirium, Delirium cordis, Fibrillar contraction, Intervermiform movements, etc.

During the last two years I have performed a large number of experiments bearing upon this subject. My earlier investigations were pursued in the physiological laboratory of University College, London, and the more recent ones in the physiological laboratory of the University of Aberdeen. I have studied the phenomena in question in the hearts of the dog, cat, rabbit, rat, mouse, hedgehog, and fowl, both in the young animal and in the adult.

The experiments were all conducted on completely anaesthetised animals; artificial respiration was carried on, a cannula being inserted in the trachea; the thorax was opened in many cases and the heart laid bare; the temperature of the animal was kept up by means of a warm pan.

I shall briefly state the main facts in my investigation.

I. The state of arhythmic fibrillar contraction is essentially due to certain changes occurring within the ventricles themselves. It is not due to the passage of any abnormal nerve impulses to the ventricles from other parts, or to the interruption of any impulses normally transmitted

*J. Physiol. 8: 296-310, 1887.

[1]Zeitschrft. f. rat. Medicin, 1850, vol. ix.

to the ventricles and necessary for their normal co-ordinated action The condition is not due to injury or irritation of the nerves that pass over the ventricles from the base of the heart

The ventricles contain within themselves the entire mechanism necessary for the execution of regular co-ordinated beats They are not dependent for this power on any nervous or mechanical connection with other parts The continuity of the nerves that pass from the auricles to the ventricles is not at all essential for the execution of regular and effective beats by the ventricles, nor is the mechanical connection between those parts necessary This is obvious from the fact that when a section is made through the auriculo-ventricular groove so as to separate the ventricles entirely from the auricles, the isolated ventricles can still exhibit their co-ordinated rhythmic contraction Instead of cutting off the ventricles, Wooldridge[1] and Tigerstedt[2] physiologically disconnected the ventricles from the auricles so as to destroy all vital connection between them while the parts were still kept *in situ* and the flood of blood through the cavities of the heart was allowed to go on, the ventricles went on beating in regular fashion though at a slower rate than before I have frequently performed a similar experiment and have watched the ventricular action as it went on, strong and regular for prolonged periods It is evident that neither the nervous, nor the mechanical connection between the auricles and the ventricles is necessary for the effective contraction of the latter It is clear that a mere solution of the continuity of the nerves passing to the ventricles does not destroy the character of the ventricular beat, and it is plain, that such a solution of continuity cannot be the cause of a sudden replacement of the normal systole by the arhythmic fibrillar form of contraction

Nor is the fibrillar contraction due to irritation of those ventricular nerve trunks Many observers have noticed its occurrence when the nerve trunks on the surface of the ventricles were being stimulated But such results appear to be due entirely to an escape of the exciting current to the underlying ventricular substance For when the nerve trunk is isolated for some little distance and precautions are taken to prevent an escape of the current, I have never found the nerve stimulation to have any effect at all in inducing the fibrillar contraction Moreover, an interrupted current readily brings about the arhythmic fibrillar condition when applied to regions of the ventricles where there are no nerve trunks, e g, to the very apex of the heart Even mechanical or thermal stimulation applied to this region may lead to the same result

The arhythmic fibrillar contraction is undoubtedly a phenomenon depending on changes within the ventricular substance, it can occur quite independently of any mechanical relation of the ventricles to the rest of the heart and of any nervous relation of the ventricles to the rest of the

[1] Arch f Anat u Physiol 1883
[2] Arch f Anat u Physiol 1884

heart or to the extra-cardiac nerves The isolated ventricles whether in the quiescent state or beating rhythmically, can by the application of faradic currents be readily thrown into the characteristic fibrillar state, just like the ventricles of an intact heart And in the intact heart the fibrillar contraction appears to be entirely uninfluenced by nerve excitation of any kind, stimulation of the vagus or any other nerve appears to produce no effect whatever

Further, the fibrillar contraction can be propagated from one part of the ventricular substance to another quite independently of the nerve trunks For if a number of overlapping incisions be made across the long diameter of the ventricles so as to leave the apex attached to the rest of the ventricles by a zig-zag isthmus of tissue, it often occurs that fibrillar movement excited by faradisation in the apex travels along the zig-zag isthmus of connecting substance, and so comes to pervade the whole of the ventricular tissue

II The arhythmic fibrillar contraction is not necessarily dependent on the destruction or paralysis of a co-ordinating centre located in any particular part of the ventricles

Kronecker and Schmey[1] succeeded in throwing the ventricles of the dog's heart into the state of fibrillar movement by piercing with a needle a certain limited part of the ventricular septum near the junction of its upper and middle thirds This result these investigators attributed to the destruction of a centre located in that region, and normally presiding over the co-ordination of the ventricular muscle in the execution of its regular beat

There is conclusive evidence that all cases of fibrillar contraction of the ventricles cannot be explained by such a hypothesis—the destruction of a co-ordinating centre localised as indicated above The fact that recovery may take place—that the ventricles may resume their co-ordinated rhythm, controverts the idea of the actual destruction of a centre essential for co-ordination Such recovery I have witnessed in several instances in the dog's heart, and in a very large number of instances in the hearts of other animals (cat, rabbit, rat, mouse, hedgehog, and fowl) Recovery occurs with different degrees of facility in different animals and in different conditions in the same animal In the dog, recovery occurs with much difficulty and only after the fibrillar contraction has lasted for a considerable space of time, indeed there very frequently is no recovery apparent—the ventricles may not recommence beating after the inco-ordinated quivering movement has ceased At times, however, a number of regular beats are seen after the termination of the fibrillar contraction A depression of the excitability of the ventricular tissue often appears to favour recovery

[1]Sitzungsber d Berliner Acad 1884

In most mammals, recovery commonly occurs. Very often it is possible to induce the fibrillar movement again and again, complete recovery occurring in the intervals, when the normal systoles are seen. In young mammals, foetal or after birth, recovery appears to be the rule; the fibrillar movement is only a temporary condition, and soon gives place to normal beats.

In birds also I have frequently observed complete recovery. The fibrillar condition is readily induced by faradisation. The ventricles exhibit the characteristic quivering movement; they become dilated with blood. In consequence of the stagnation of blood in the ventricles, the auricles also become gorged and may become so over-distended that they temporarily stop beating; asphyxial convulsions occur in the skeletal muscles. After a time, however, the fibrillar movement ceases, the ventricles remain quiescent for a little time, then give a regular co-ordinated beat and the action of the whole heart proceeds in the normal fashion. These phenomena can by the application of a current of the proper strength be induced again and again.

Further, in addition to the evidence afforded by the recovery of the ventricular beat, there is the fact that the arhythmic fibrillar movement may very readily be induced by means that are not capable of destroying a deep-seated co-ordinating centre, e.g., faradic, mechanical, or thermal stimulation of the surface of the ventricles even at the very apex.

Since it is certain that the arhythmic fibrillar movement is not necessarily due to the actual destruction of a co-ordinating centre, there next arises the question as to whether the fibrillar contraction may be due to the temporary paralysis of such a centre as that indicated by Kronecker—of the existence of which no histological evidence has, as far as I am aware, been advanced.

I shall at a later stage of this paper have to adduce some evidence regarding the action of certain poisons which when injected into the blood lead to the occurrence of fibrillar contraction of the ventricles. Such a result might be regarded as due to the paralysis of a hypothetical co-ordinating centre. And the fibrillar contraction caused by stimulation (electrical, mechanical, etc.) of the ventricular surface might be explained in a somewhat similar fashion. For it is conceivable that such stimulation might give rise to strong abnormal afferent impulses with the result of deranging or paralysing the action of the co-ordinating centre; the paralysis might be a temporary one or might be permanent according to the particular circumstances in each case.

But there is strong evidence against the adoption of such a view—against the idea that the phenomena are due to the behaviour of a definite co-ordinating centre localised above the middle of the ventricular septum in the dog's heart. For the influence of such a centre does not appear to be at all essential for the production of co-ordinated and efficient beats

The amputated apex—the lower third or fourth of the ventricles—both in the dog and in all other mammals I have examined,—is capable of executing co-ordinated beats when it is entirely removed from all possible relation with any co-ordinating centre high up in the ventricular septum This one can verify by the rough but conclusive experiment of tying the freshly removed apex of a vigorous heart upon a double cannula through which the cavity of the left ventricle can be filled with blood, the propulsion of fluid at each beat of the isolated apex can be readily observed The visible character of the beat may also be noted, and the co-ordinated nature of the contraction causing a marked diminution of the cavity at each systole may be felt with the finger tip inserted into the cavity of the left ventricle It is obvious then that the paralysis of a co-ordinating centre in the upper half of the ventricular septum would not necessarily cause a loss of co-ordination in the contraction of the whole of the ventricular muscle

Further there is the fact that the apical portion of the ventricles—capable as it is of performing regular beats—can be thrown into a state of fibrillar contraction by the usual means, e g, the application of a faradic current In the isolated apical part of the ventricles (in all the mammals I have examined) I have been able to excite the fibrillar contraction again and again, recovery occurring in the intervals, and co-ordinated beats being given in response to single stimuli applied during those intervals It appears then that the behaviour of the intact ventricles and of the entire isolated ventricles both as regards co-ordinated single beats and as regards the fibrillar contraction can be reproduced in the isolated apical portion, and hence we may conclude that these phenomena are not necessarily dependent on the condition of any co-ordinating centre in the upper half of the ventricles

III The outstanding features of the arhythmic fibrillar contraction are (1) the complexity of movement, (2) its persistence, (3) its rapidity

The complexity of the fibrillar movement appears to be in direct relation to the complex arrangement of the muscular fibres of the ventricular walls

In the ventricles we have bundles of muscular fibres forming by their interlacement a texture of remarkable complexity It appears that the complex quivering movement depends on the passage of rapidly repeated waves of contraction along the complexly arranged muscular bundles which are enclosed by connective tissue and joined to one another by cross-branches It is readily conceivable that contractions simply conducted along the muscular fibres should be transmitted with unequal rapidity along the ventricular walls and should reach the same part of the ventricular wall at different points of time Some bundles of fibres are in a state of contraction while neighbouring bundles are relaxed

and so instead of a co-ordinated contraction causing a definite and (in the case of the left ventricle) concentric narrowing of the ventricular cavity, there occurs an irregular and complicated arhythmic oscillation of the ventricular walls which remain in a position of diastole

That the complexity of the fibrillar movement in the grown animal depends on the character of the muscular structure is illustrated by the appearances presented by the corresponding movements in the hearts of foetal and young animals In these as long as the structure of the ventricles is simple the rapid movement excited by faradisation is of a simple character And just as the complexity of the muscular structure increases in the growing animal so does the complexity of the movement obtained There can be observed a complete gradation from the simple movement excited by faradisation in the ventricles of the mammalian foetus or of the chick (a movement much resembling that seen in similar circumstances in the comparatively simple ventricles of cold-blooded animals) to the very characteristic and striking complexity of the fibrillar contraction in the adult mammal or bird It is obvious that the nature of the muscular structure is a cardinal feature, and it is not very evident why such should be the case if the condition is due to derangement of a nervous mechanism causing it to discharge irregularly, for a deranged nervous mechanism discharging irregularly might cause an equally irregular movement whether the muscular arrangement is simple or complex

The simpler character of the movement excited by faradisation in the auricles of warm-blooded animals is probably due to the simpler histological structure of the auricular walls and the simpler mode of propagation of the normal contraction

The persistence of the fibrillar contraction appears to depend on the high excitability of the ventricular tissue

When the fibrillar contraction has been brought about by stimulation of the ventricles, the prolonged continuance of the movement, after the cessation of the exciting cause is a striking feature It appears to be a result of the excitation of a highly excitable, and probably highly rhythmic tissue The duration of the movement varies in each instance with the excitability of the ventricular muscle It can easily be shown, that in certain depressed conditions of the ventricular tissue, the duration of the fibrillar movement, induced by stimulation, is much diminished and when the ventricular excitability is very much lowered, (by gradual cooling exhaustion etc) it frequently occurs that the fibrillar contraction does not persist after the stimulating current is discontinued, it simply occurs during the passage of faradic current and passes off at the cessation of that current Indeed, in some instances it may be found that the fibrillar contraction cannot be excited at all by faradisation, whilst the ventricles are still capable of executing single beats A cer-

tain degree of excitability is necessary for the production of the fibrillar contraction in response to stimulation

Similar facts with reference to the duration of movement, after the discontinuance of the exciting cause, may be seen in the hearts of cold-blooded animals In the heart of the eel, for example, where there are a number of parts possessed of different degrees of excitability and rhythmic power, very marked differences are to be observed in the behaviour of the several parts after a stimulating current has been temporarily applied The sinus with the basal wall, and the canalis auricularis, the auricle, and the ventricle, form a descending series as far as rhythmic power is concerned and they present similar differences as regards the after effects of stimulation In the ventricle a short period of moderate stimulation excites a movement, which usually terminates immediately or very soon after the end of the stimulation, the precise period at which the movement terminates varies according to the strength of the exciting current and the excitability of the ventricle, in a very excitable ventricle (in situ, with the normal circulation intact) the movement may persist for some little time after the stimulation has ended In the auricle, the movement usually persists longer, and in the sinus a great deal longer still Indeed, in the sinus a single stimulation can often lead to a series of beats, whereas in the case of the auricle and still more in the ventricle a single stimulation excites but a single contraction Moderate heating of the tissue causing a rise in its excitability usually leads to a marked increase in the persistence of the movement excited by a short period of stimulation

Similarly in the mammalian heart the duration of the fibrillar movement after the end of the period of excitation varies In the foetal heart it lasts but a short time, and in adult hearts that have been much depressed by exhaustion and by gradual cooling, the fibrillar movement usually passes away very much earlier than it does in a more excitable heart

The mechanism of the movement, as will be subsequently stated, appears to be such as to involve its continuance as long as the excitability of the ventricular tissue is sufficiently high

The cause of the great rapidity of the series of contractions that course over the ventricular fibres during the state of fibrillar contraction will be considered later on

IV The arhythmic fibrillar contraction is in one class of cases a phenomenon of irritation induced by the action of various recognised stimulants

The state of excitement generated in the muscular tissue appears to resemble in some respects the state of excitement obtaining in the nerve cells of the cortex cerebri during an attack of epileptiform convulsions induced by strong stimulation

It has been stated that the duration of the fibrillar contraction depends on the excitability of the ventricular tissue In like manner the readiness with which the fibrillar contraction can be excited by stimulation, is in close relation with the ventricular irritability In a depressed heart it is frequently very difficult to produce the phenomenon in question by stimulation, very powerful currents are necessary

On the other hand, when the excitability is heightened, it is easy to induce the fibrillar contraction The occurrence of this phenomenon in response to stimulation is retarded and its duration shortened by conditions that depress the excitability of the cardiac muscle its occurrence is favoured and its duration prolonged by causes that augment the cardiac irritability In an exhausted heart it can frequently be seen that faradisation of the right ventricle leads to the occurrence of the fibrillar contractions in both ventricles, when such a result has ceased to be obtained by faradisation of the left ventricle The difference in the behaviour of the ventricles, in this respect appears to be due to the greater persistence of the excitability in the right ventricle as compared with the left

When the fibrillar contraction has been excited by stimulation it can often be arrested by the cautious application of depressant measures calculated to diminish the excitability of the ventricular tissue, e g, deprivation of blood supply and cooling

The readiness with which the ventricles are thrown into the fibrillar condition varies remarkably in different conditions of the cardiac tissues In a normally contracting and vigorous heart it usually requires a faradic current of considerable strength to produce the result in question And it is not easy in these circumstances to induce the fibrillar contraction by mechanical or thermal stimulation But in certain changed conditions of the organ it becomes extremely easy to throw the ventricles into the fibrillar movement An exceedingly weak faradic current, a touch with a hot wire, a mere scratch with the point of a pin, slight friction of the ventricles against the cut end of a rib, or even slight pressure with the finger are each of them sufficient at such times to excite the fibrillar contraction The precise conditions in which there is such a remarkable sensitiveness to certain forms of stimulation are difficult to define, I have frequently observed such a sensitiveness when the action of the heart has been deranged or impaired by various causes—among others by a temporary arrest of the respiration or by a great fall in the blood pressure leading to anaemia of the cardiac tissues, et cetera, the phase of increased sensitiveness seems to be a transitory one

The frequent occurrence in the ventricles of such phases of extreme readiness to assume the fibrillar form of contraction appears to me to be of great importance with regard to the question of electrical stimulation of the heart in man during sudden cardiac failure (syncope during

the administration of anaesthetics, etc.) It is obvious that the use of faradic currents of any strength is attended with grave danger in such cases. For although Von Ziemssen and others have applied the induced current to the human heart without any serious results, the conditions were different in such cases. They experimented with normally beating hearts, the tendency of which to assume the fibrillar form of contraction is strikingly less than what frequently obtains in hearts placed in abnormal circumstances—necessarily present in those cases where the faradic current is employed clinically.

But although the exposed heart in the opened thorax may be readily thrown into the arhythmic fibrillar contraction by faradisation, it may be urged that possibly the normally beating heart in the intact thorax, is not similarly affected. I have on several occasions introduced a fine platinum wire electrode through the chest wall so as to come in contact with the ventricles and have then faradised, the other electrode being applied to the outside of the chest wall, the fibrillar contraction was at once induced.

By the use of single induction shocks I have never seen the fibrillar contraction excited either when the shock is passed through the thoracic walls or when it is applied to the exposed heart. The single induction shock seems to be free from the dangers accompanying the use of the faradic current. Hence I have urged its superiority as a means of cardiac stimulation, in a paper to be read at the Ninth International Medical Congress at Washington.

The extreme readiness with which in certain circumstances the ventricles are thrown into the fibrillar contraction by any form of irritation, mechanical as well as electrical, renders it apparent that the experiment of puncturing the heart in order to destroy a certain part is attended with many difficulties. For very frequently the mere mechanical irritation would be amply sufficient to produce all the phenomena usually resulting from faradisation. And this condition of increased sensitiveness to irritation and increased tendency to assume the fibrillar mode of contraction appears to occur with special frequency and to a very marked degree in the heart of the dog.

V. In another class of cases the fibrillar contraction is induced by the more or less sudden action of certain influences of a depressing nature.

The injection of certain salts (e.g., bromide of potassium in strong solution) into the blood appears to induce the fibrillar condition in a very short space of time (frequently within one minute). A dose of about 0.1 gramme is sufficient in the hedgehog.

When such an injection is made (cat and hedgehog) there is almost immediately a marked change in the character of the systole. The origin and course of the contraction become very apparent both in the auricles and in the ventricles. In the former it passes forwards from the entrance

of the great veins, in the latter it sweeps from the base of the heart towards the apex, on the front of the heart the contraction can be most distinctly seen beginning at the conus arteriosus and passing downwards. The ventricles become dilated with blood, the contractions are evidently unable to empty the cavities. When the heart is in a depressed state no further important change may be observed, the contractions gradually become weaker and slower until they cease altogether. But in the case of a vigorous heart there usually occurs a striking change—a short time after the injection of the bromide. The ventricles go into the state of fibrillar contraction with its usual features.

I have not as yet seen any complete recovery from the inco-ordinated condition produced in this way. The ventricles do not seem to recover their power of giving regular beats. Single contractions may occur after the rapid quivering movement has ceased but they appear to be fibrillar in their nature. And any contractions excited by single induction shocks in such circumstances appear to be of the same character.

After the injection of a solution of atropin I have observed somewhat similar phenomena, here, however the fibrillar movement was arrested by the injection of pilocarpin and complete recovery of the ventricular beat took place.

I have on some occasions observed phenomena of the same kind when an animal (cat) was suddenly and powerfully cooled by the application of a mixture of ice and salt to the surface of the skin and the insertion of an ice bag into the abdominal cavity. After the cooling had gone on for a time, the ventricles suddenly passed into the state of fibrillar contraction.

See and others have described the occurrence of a similar fibrillar movement in the dog's ventricles as one of the results of sudden occlusion of the coronary arteries.

VI. The arhythmic fibrillar contraction is fundamentally different from a rapid series of normal contractions. Its genesis probably assumes in all cases one or other of two forms.

It is probable that the normally contracting ventricles possess within themselves certain co-ordinating arrangements in virtue of which the muscular contraction constituting a normal beat rapidly traverses the whole of the ventricular substances, causing a uniform or nearly uniform contraction of all the fibres of the ventricular walls thus leading to a concentric narrowing of the ventricular cavity and a consequent expulsion of its contents. The co-ordinating arrangements appear to exist in the lower portion of the ventricles as well as in the upper portion, for it has been seen that the apical part can execute co-ordinated beats when severed from the rest of the heart.

A normal co-ordinated contraction appears to be essentially different from the individual beats that may be seen after poisoning with bromide

of potassium and occasionally in other conditions In the latter case the contraction is obviously of a peristaltic nature, the contraction wave can be seen passing over the ventricular surface in definite directions The contraction may be caused to start at any part in the ventricular substance by the application of a single direct stimulus, the contraction begins in the stimulated area and hence spreads over the rest of the ventricles, a phenomenon precisely similar to what one sees in the hearts of cold-blooded animals

The peristaltic contraction evidently passes over the various interlacing bundles at different points of time, so that the whole thickness of the ventricular wall at any part is never uniformly contracted Hence there is a wiry feel distinctly perceptible when the ventricles are held between the fingers as the peristaltic contraction is passing through its substance, certain fibres are hardened by the presence of contraction in them while neighbouring fibres are relaxed and soft Such peristaltic contraction appears to be incapable of emptying the ventricular cavities of their contents, *it appears to be essentially different from a co-ordinated beat however slow the latter may be* A co-ordinated beat never presents a wiry feel to the finger, it gives the sensation of a steady and uniform hardening of the muscle substance—of precisely the same nature as the hardening one feels in a skeletal muscle during its contraction The contraction seems to involve as a whole the complicated interlacement of fibres forming the ventricular wall

It appears then that the ventricles are capable of executing two forms of beat One is the co-ordinated contraction seen in the normal heart and capable of being excited by artificial stimulation (e g, by single induction shocks) either in an intact heart, or in the fresh and vigorous excised ventricles or ventricle-apex The other form of beat is the inco-ordinated or simple peristaltic contraction, such as may be seen after poisoning with bromide of potassium and in certain other conditions

VII The state of arhythmic fibrillar contraction (delirium cordis, etc) appears to be constituted by a rapid succession of inco-ordinated peristaltic contractions—a condition that can be brought about either (1) by the influence of certain depressing or paralysing agents upon the ventricular tissue, or (2) by the application of certain forms of stimulation to the ventricular tissue

In the first class of cases the depressing influences alluded to probably throw out of gear the co-ordinating arrangements while they leave the muscular irritability intact—or it may be even augmented largely Then the excitable (and probably highly rhythmic) muscle contracts, but its excitation instead of assuming the form of a normal beat becomes a peristaltic contraction wave along the complexly arranged and intercommunicating muscular bundles And if the ventricular muscle is in an excitable state there would naturally occur a rapid series of such inco-

ordinated peristaltic contractions For apart from the possibility of rapid spontaneous discharges of energy by the muscular fibres, there seems to be another probable cause of continued and rapid movement The peristaltic contraction travelling along such a structure as that of the ventricular wall must reach adjacent muscle bundles at different points of time and since these bundles are connected with one another by anastomosing branches, the contraction would naturally be propagated from one contracting fibre to another over which the contraction wave had already passed Hence if the fibres are sufficiently excitable and ready to respond to contraction waves reaching them, there would evidently be a more or less rapid series of contraction in each muscular bundle, in consequence of the successive contraction waves reaching that bundle from different directions along its fibres of anastomosis with other bundles Hence the movement would tend to go on until the excitability of the muscular tissue had been lowered so that it failed to respond with a rapid series of contractions Then there might be some isolated peristaltic contractions such as I have often seen after the cessation of the fibrillar movement

In the second class of cases—when the fibrillar contraction is excited by stimulation (e g, faradisation of the surface of the ventricles) there appears to be a condition of violent excitement set up in the muscular tissue The excitation of the muscular fibres travels peristaltically, producing the characteristic movement the inco-ordinated contraction of the various fibres may be most distinctly realised when the ventricles are held between the forefinger and thumb, there is a sort of wriggling sensation to be felt as the individual muscular bundles become hard and wiry while the contraction is passing over them in succession The co-ordinating arrangements of the ventricles are powerless to regulate and guide the contractions, those co-ordinating arrangements are very possibly not paralysed nor rendered incapable of action, but they are temporarily superseded and rendered inoperative by the excessive state of excitement which pervades the muscular fibres—just as the cerebrospinal co-ordinating mechanism might be rendered impotent by strong local stimulation of the skeletal muscles When the fibrillar movement having become less rapid has at length stopped—its duration depending on the excitability of the muscle—there ensues a pause

Then there may be a recovery of the normal co-ordinated beat provided the fibrillar condition (and consequent blood stasis) has not lasted so long as to involve a paralysis or death of the co-ordinating mechanism

When the last mentioned change has taken place, any beats that may occur are of the fibrillar character

VIII The phenomena resulting from faradic stimulation of the auricles differ in various respects from those seen in the ventricles

The application of the current sets the auricles into a rapid flutter, the rapidity of which largely depends upon the excitability of the auricular

tissue and the strength of current employed. The movements are regular, they seem to consist of a series of contractions originating in the stimulated area and thence spreading over the rest of the tissue. The movement does not show any distinct sign of inco-ordination, it looks like a rapid series of contraction waves passing over the auricular walls. The difference between this appearance and that seen in the ventricles probably depends on the simpler structure and arrangements obtaining in the auricles.

The persistence of the movement after the discontinuance of the stimulating current varies according to the excitability of the auricular tissue and strength of current employed. In very excitable conditions the rapid movement lasts for a considerable time, in depressed states the movement ceases almost immediately after the stimulation has ended. The persistence after the use of a strong current is, *caeteris paribus,* usually very much greater than when a weak current has been employed to excite the fluttering action.

IX. The movements excited by faradisation in the auricles and ventricles differ very markedly in their relation to the inhibitory influence of the vagus nerve. The fibrillar movement in the ventricles appears to be entirely unaffected by vagus stimulation, the fluttering movement of the auricles can be checked or arrested by the influence of the vagus.

Sometimes, when the auricles are very excitable, the fluttering movement is entirely suspended during vagus stimulation only to reappear when the inhibitory influence has passed away. The vagus influence appears to act by weakening the individual contractions to the point of invisibility. At other times the contractions are markedly weakened without being rendered invisible. Often the movement is entirely arrested and does not recur, the normal action of the auricles goes on after the period of inhibition has passed.

The relation of the vagus nerve to the auricular muscle seems to be entirely different from the relation of that nerve to the ventricular muscle.

1888

GRAHAM STEELL

DESCRIPTION OF THE MURMUR OF HIGH PRESSURE IN THE PULMONARY ARTERY, LATER TO BE KNOWN AS THE GRAHAM STEELL MURMUR

GRAHAM STEELL

(1851- —)

We regret our inability to include in this volume a biographic sketch of Dr Graham Steell Dr Steell has granted us permission to reproduce his paper on "The Murmur of High-Pressure in the Pulmonary Artery," but he has requested that we do not attempt to delineate the leading incidents in his life and career We are happy to record that Dr Steell is still living He was born on July 27, 1851

THE MURMUR OF HIGH-PRESSURE IN THE PULMONARY ARTERY*

By

GRAHAM STEELL, M.D

Assistant Physician to the Manchester Royal Infirmary

THERE has long been question among physiologists of a safety-valve action of the tricuspid valves Few clinicists will deny that, in disease, a similar occurrence takes place on the left side of the heart, the mitral valves becoming incompetent when the left ventricle is embarrassed under the effort which it is called upon to make The muscle-element in the valve apparatus of the auriculo-ventricular orifices must be borne in mind in this relation, for it is by interference with muscle-action that incompetence of the valves is secured, and relief temporarily afforded in both cases It is, therefore, the important part played by the heart-muscle in the establishment and maintenance of closure of both the tricuspid and mitral valves which renders possible the sudden production of their incompetence under special circumstances In the case of the mitral valves, the causes which demand regurgitation in the way indicated have been, as a rule, long at work, and the accomplishment of regurgitation has been preceded by a series of changes The valve-apparatus of the great arteries of the heart, unlike that of the auriculo-ventricular orifices, is independent of muscle-action, so that an analogous safety-valve action, in its case, appears to be out of the question In health I believe it to be so, and, at the same time, I do not hesitate to express my disbelief in the rupture of a sound valve In disease it is otherwise, and the clinical study of arterial high tension, aortic dilatation, and final incompetence of the valves, forces me to the admission that the arterial valves, like the auriculo-ventricular, do, under the strain

*Med Chron Manchester, 9 182-188 1888-89

of extreme tension long continued, permit of regurgitation through them. Thus there occurs an action analogous to a safety-valve one, although the name is less appropriate, since there is no threatened asystole to be obviated in their case, as there is in that of the mitral valves, inasmuch as the recoil or systole, of the elastic arteries is not a vital action It is not my purpose here to discuss aortic regurgitation arising from dilatation, and to trace its origin directly and apart from induced disease of the valves, to high arterial tension I wish to plead for the admission among the recognized auscultatory signs of disease of *a murmur due to pulmonary regurgitation, such regurgitation occurring independently of disease or deformity of the valves, and as the result of long-continued excess of blood pressure in the pulmonary artery*

In cases of mitral obstruction there is occasionally heard over the pulmonary area (the sternal extremity of the third left costal cartilage), and below this region for the distance of an inch or two along the left border of the sternum, and rarely over the lowest part of the bone itself, a soft blowing diastolic murmur immediately following, or, more exactly, running off from the accentuated second sound, while the usual indications of aortic regurgitation afforded by the pulse, etc, are absent The maximum intensity of the murmur may be regarded as situated at the sternal end of the third and fourth intercostal spaces When the second sound is reduplicated, the murmur proceeds from its latter part That such a murmur as I have described does exist, there can, I think, be no doubt Let me quote, with regard to it, the testimony of my revered master, Dr G W Balfour, though he gives a very different explanation of the murmur from that which I advocate Speaking of the rare occurrence of pulmonary incompetence from disease of the valves, he says * "I mention it just now mainly for the purpose of warning you against being led into mistaking an auricular diastolic murmur for a pulmonary diastolic one I have already pointed out that mitral stenosis is not infrequently associated with a diastolic murmur apart and distinct from its own peculiar presystolic murmur Now and then this diastolic murmur of auricular origin has its position of maximum intensity at the sternal end of the fourth rib, a position in which it might readily be mistaken for a pulmonary diastolic murmur, and possibly has been so mistaken " In another place, he speaks of the position of maximum intensity being "frequently in the pulmonary area "† I must here remark, that the murmur, which I have described, is altogether different from the obstructive diastolic murmur of mitral stenosis, which is essentially an apex murmur, and, moreover, is wanting in the soft blowing quality of the pulmonary regurgitant murmur The mitral murmur, too, runs off from the first part of a reduplicated second sound, the pulmonary from the last part

*Clinical Lectures on Diseases of the Heart and Aorta, p 218
†Note, p 119

I am prepared for the objection that the murmur under consideration is only the murmur of a slight amount of aortic regurgitation, the usual evidence of which in the pulse is masked by the mitral lesion. How difficult it is to distinguish between the murmurs of aortic and pulmonary regurgitation respectively, by means of auscultation alone, will be admitted when it is remembered that the ordinary murmur of aortic regurgitation is probably conveyed to the surface directly through the right ventricle and especially through that part of it which is called the conus arteriosus or infundibulum. With reference to this fact, the late Doctor Gibson's description is interesting. He says:* "The root of the aorta, including its orifice, valve and sinuses occupies the space between the pulmonic and tricuspid orifices. The root of the aorta and the aortic vestibule, which is the channel or chamber with rigid walls that leads to it from the cavity of the left ventricle, project forwards in front of that cavity and of its mitral orifice, so that the orifice of the aorta, covered by the posterior wall of the conus arteriosus, interposes itself, as has just been stated between the pulmonic and tricuspid orifices. By this arrangement the aortic orifice advances more nearly to the front of the chest, the shallow conus arteriosus being in front of the orifice, and the deep cavity of the right ventricle being below it. Hence the murmur of aortic regurgitation, and an intensified aortic second sound, and coincident doubling of that sound, are heard loudly over and to the left of the middle third of the sternum in front of the arterial cone and the root of the aorta." When there is pulmonary regurgitation, again the conus arteriosus will be the seat of the fluid veins which produce the diastolic murmur, and over it, accordingly, we may expect to find the maximum intensity of the murmur. The fact that in mitral stenosis the conus arteriosus is generally enlarged, and therefore extends to the left of the sternum to a greater degree than in the normal condition, does not seem to offer an entirely satisfactory explanation of the peculiar localization of this murmur, supposing it to be an aortic regurgitant murmur, almost wholly to the left of the sternum. I must admit, however, that in cases in which a diastolic murmur was audible over the lower half of the sternum, and the other evidence in favor of aortic regurgitation was strong, I have often been struck by the loudness of the murmur to the left of the sternum. Possibly enlargement of the right ventricle, with extension of the conus arteriosus to the left, was present in all these cases.

The murmur of high-pressure in the pulmonary artery is not peculiar to mitral stenosis, although it is most commonly met with, as a consequence of this lesion. Any long-continued obstruction in the pulmonary circulation may produce it. The pulmonary valves, like the aortic, do not readily become incompetent, apart from structural change. Probably no

*Reynold's System of Medicine. Vol. IV. p. 84.

amount of blood pressure in the pulmonary artery will render them so suddenly, as, at least, theoretically, the mitral valves may be rendered incompetent. Changes in the vessel, with widening of its channel, and, eventually, of its orifice, long precede the occurrence of incompetence of its valves. The pulmonary murmur of high-pressure is probably never persistent at first, and one of its most remarkable features is, as a rule, its variableness in intensity. On some days it will be distinctly heard, on others, it will be indistinct, or even inaudible, while extreme accentuation of the pulmonary second sound is always present, the closure of the pulmonary semilunar valves being generally perceptible to the hand placed over the pulmonary area, as a sharp thud. This non-persistence of the murmur, in the earlier stages, at any rate, is only what the study of dilatation of the aorta and the consequent regurgitation would lead us to expect. Indeed, so common is a soft, blowing murmur, after an accentuated aortic second sound, that extreme accentuation should make us listen, with special care, for a murmur, and even though it be absent on the first occasion the search should not be abandoned. My belief is, that when the aortic second sound is extremely accentuated, regurgitation, to some extent, will probably occur sooner or later. Its supervention in aneurism of the first part of the arch of the aorta is a familiar fact. Post mortem, enlargement of the left ventricle, in these cases, may be a better indication of regurgitation having occurred during life than the usual test of filling the cut aorta with water, a proceeding which cannot imitate the action of the forcible blood-currents in the living body. An accentuated second sound is no way incompatible with a certain amount of incompetence of the semilunar valves; on the contrary, an accentuated second sound, associated with a regurgitant murmur, is clinically common.

Writing in 1881,* after describing the regurgitant murmur of aortic dilation, I referred to the murmur which is the subject of this paper, as follows: "I am inclined to believe that a murmur of similar mechanism occurs on the right side of the heart, when there is much obstruction to the pulmonary circulation, with a dilated pulmonary artery." My subsequent experience has only served to confirm the opinion thus cautiously expressed more than seven years ago, though my faith has from time to time been shaken by a case presenting a murmur which I had at first imagined to be an example, but which, on further investigation, proved to be of aortic origin.

At a meeting of the Clinical Society of London,† on January 27th, 1888, Sir Dyce Duckworth related a case of "tricuspid and mitral stenosis in which physical signs of pulmonary arterial reflux were present," and stated his belief that the pulmonary arterial reflux was probably

*The Physical Signs of Cardiac Disease, Maclachlan and Steward, Edinburgh, 1881.
†British Medical Journal, February, 1888.

explicable by the dilated state of the vessel which was found after death In this case the murmur was not persistent, in this respect corresponding to the description given above

Only a few cases of incompetence of the pulmonary valves from structural change or deformity are on record One of these was related by the late Dr J Warburton Begbie, who described the site of the murmur in the following words "On more careful examination, the thrill was found to be almost entirely limited to the situation in which a loud systolic murmur was heard with the greatest degree of intensity That was at the left border of the sternum, over the cartilage of the third rib The systolic murmur thus distinguished was blowing in character and of unusual loudness, in the same situation it was followed by a diastolic murmur of much less intensity The systolic murmur was readily distinguished over the whole upper part of the chest, but with much facility the seat of its greatest intensity was determined to be that already indicated The diastolic murmur was limited, or almost limited to the same situation "*

Doctor Hayden† writes —"From aortic diastolic murmur that of pulmonary inadequacy may be distinguished, not only by its seat of origin being to the left of the sternum, but likewise, as urged by Doctors Gordon and Begbie, by the absence of visible pulsations in the arteries "

Doctor Hope‡ wrote —"Diastolic murmur of the pulmonic valves I created this murmur artificially in an ass poisoned with woorara, by making a perforation through one valve We found the murmur soft, prolonged, and audible down the ventricle, exactly as in aortic regurgitation In the human subject the pulmonic would probably be louder than the aortic diastolic murmur, because its seat is nearer the surface "

Doctor Bramwell§ writes —"Pulmonary incompetence is attended with a diastolic murmur, which has its point of differential maximum intensity in the pulmonary area, and its direction of propagation downwards to the right The murmur, like the diastolic murmur of aortic regurgitation, would probably in many cases, be best heard at the lower end of the sternum "

The limitation of the murmur of pulmonary regurgitation to the left of the sternum does not seem to be in accordance with theoretical considerations, for we should expect the murmur to be conducted, as Doctor Bramwell says, "downwards and *to the right,*" over the right ventricle Apparently this is not the case, as a rule, and when the murmur is conducted to the lowest part of the sternum its maximum intensity will always be found to the left of the bone

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*Beale s Archives of Medicine Vol II page 11
†Diseases of the Heart and Aorta p 1 005
‡A Treatise on the Diseases of the Heart and Great Vessels 3rd edition p 76
§Diseases of the Heart and Thoracic Aorta

With regards to prognosis, the murmur probably points to the vigour of the right ventricle being well maintained

The discrimination of the murmur in question from the murmur of aortic regurgitation will always be a matter of peculiar difficulty, from the reasons already indicated, but it is surely of some importance that conditions so dissimilar as arterial regurgitation on the right and on the left sides respectively of the heart should be distinguished at the bed side

In matters medical there should not even be the semblance of special pleading Let me, therefore, in conclusion, distinguish between the facts observed, and the theory advocated to explain them It is simply a matter of experience, that in a considerable proportion of typical cases of mitral stenosis there is audible a murmur with the characters and in the situation described above, while in the same cases the left ventricle is not enlarged* and the pulse does not possess the peculiarities which we know to be produced by aortic regurgitation Shall we explain the fact by saying that the mitral stenosis prevents the enlargement of the left ventricle, which would otherwise result from aortic regurgitation, and at the same time interferes with the due development of Corrigan's pulse, and moreover that in these cases the leakage through the aortic valves is trifling in amount? We may do so plausibly enough Or shall we agree with the late Doctor Fagge, whose every sentence deserves attention, in his statement with reference to a case of pulmonary regurgitation from disease of the valves, in which case the great rarity of the disease led to its rejection as a diagnosis † "Indeed one can hardly expect in future to attain to greater accuracy, for (as we shall presently see) the pulse may fail to be characteristic of aortic regurgitation even when this disease exists, and the tendency of aortic diastolic murmurs to be transmitted downwards along the sternum, must always prevent a pulmonary regurgitant murmur from being identified by its being heard over the right ventricle," and confess that, under the circumstances, we cannot distinguish an aortic from a pulmonary regurgitant murmur? To those to whom neither the dogmatic assertion that all such murmurs as I have described, are, in spite of the absence of any confirmatory evidence, aortic in origin, nor the sceptical *non possumus* of Doctor Fagge, commends itself, I would urge a careful consideration of "the high-pressure murmur of the pulmonary artery" as a feasible explanation I have stated my own opinion, from others I ask only that this murmur should find a place—however subordinate—among the physical signs of disease, which they recognise

*We occasionally meet with an enlarged left ventricle in a case of apparently *pure* mitral stenosis The cause of such enlargement was long a mystery to me, but I now believe that it is produced at an early period of the disease while mitral regurgitation was the only or predominant effect of the valve changes

†Reynolds System of Medicine Vol IV p 646

1888

ÉTIENNE-LOUIS ARTHUR FALLOT

DESCRIPTION OF THE "MALADIE BLEUE," LATER TO BE KNOWN AS THE TETRALOGY OF FALLOT

ÉTIENNE-LOUIS ARTHUR FALLOT

(1850 1911)

ÉTIENNE-LOUIS ARTHUR FALLOT was born in Cette, France, on September 29, 1850 He was educated at the Lycée at Marseille, where he received a prize for high scholastic ability He studied medicine at the École de Médecine at Marseille After graduation he served as substitute professor of medicine at the University of Marseille from 1882 to 1886 In 1886 Fallot took charge of the course of pathologic anatomy He continued in this capacity until 1888, in which year he was appointed professor of hygiene and legal medicine He continued in this post until his death on April 30, 1911

Because of Fallot's request that no eulogy be written after his death, little biographic information concerning him is available Fallot's memory is perpetuated, however, by his studies on the congenital affections of the heart He described with precision the most common anatomic type He demonstrated in 1888 that the cardiac lesions of the "maladie bleue" (morbus caeruleus) may be summed up in a characteristic tetralogy which has since been called by his name We are reproducing in translation Fallot's summary of this important work

CONTRIBUTION TO THE PATHOLOGIC ANATOMY OF MORBUS CAERULEUS (CARDIAC CYANOSIS)*

By

DR. A. FALLOT

Here we conclude the presentation of the results of our investigations, not that we pretend to have covered the subject completely, or to have discussed all of its details, but because we have reached the limit that we set at the beginning The following lines briefly summarize the conclusions which we believe may be drawn from our study

I Until now, clinicians have considered the precise diagnosis of anatomic lesions of morbus caeruleus of almost unsurmountable difficulty, as if it could be pronounced only as a vague and uncertain hypothesis On the contrary, we see from our observations that cyanosis, especially in the adult, is the result of a small number of cardiac malformations well determined

II One of these cardiac malformations is much more frequent than the others, since we have found it in about 74 per cent of our cases, this is what the clinician should diagnose and in doing so, his chances of error are relatively slight

III This malformation consists of a true anatomopathologic type represented by the following tetralogy (1) Stenosis of the pulmonary artery, (2) Interventricular communication, (3) Deviation of the origin of the aorta to the right, (4) Hypertrophy, almost always concentric, of the right ventricle —Failure of obliteration of the foramen ovale may occasionally be added in a wholly accessory manner

IV One cannot, at present, attribute cyanosis to the persistence of the foramen ovale without putting himself in explicit opposition to the great majority of observed facts, communication of the two auricles, when it exists without any other concomitant cardiac lesion, does not produce cyanosis

V From the historical point of view one finds, with the writers of the last century and of the beginning of the present one, a fair number of observations on cyanosis, for the most part they offer the interesting

*Fallot A Contribution à l'anatomie patholog[illegible] maladie bleue (cyanose cardiaque), Marseille Médical 25 418-420, [illegible] by Dr L. Morissette, Rochester, Minn

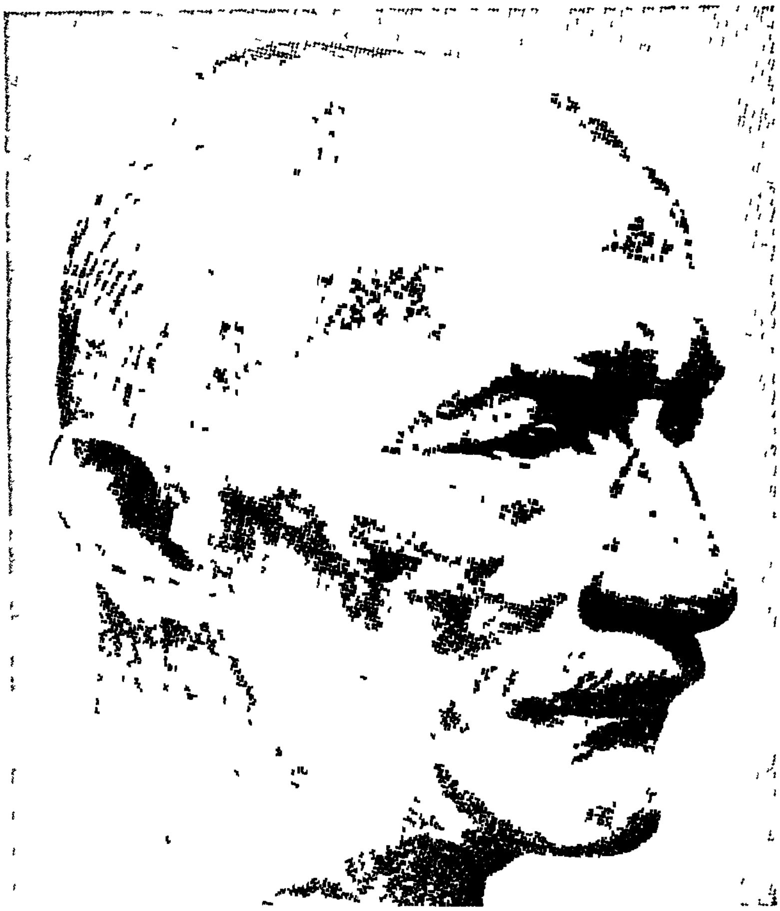
peculiarity that the existence of the various cardiac lesions previously mentioned is met with and fully described

VI Finally, from the point of view of pathogenesis, the theory which considers interventricular communication as a simple phenomenon, appertaining to a group of reversive anomalies, rests only on a superficial and inexact interpretation of facts, in persons with morbus caeruleus, the incompletely developed septum cannot by any means be considered as the analogy of the false septum of vertebrates to communicating ventricles It seems to be much more logical and more in conformity with the laws of physiology to regard the whole series of cardiac changes enumerated as wholly the result of pulmonary stenosis As to the cause of this, we believe that we must attribute it not to a simple arresting of development but rather to a pathologic process developing during intra-uterine life at the level of the pulmonary valves and of the region of the infundibulum which is contiguous to them

1893

WILHELM HIS, JR

DESCRIPTION OF THE AURICULOVENTRICULAR BUNDLE (BUNDLE OF HIS)



WILHELM HIS, JR

(Courtesy National Service Publishing Co)

WILHELM HIS, JR.

(1863-1934)

"The knowledge of the denervated beating heart and the significance of the nervous function is today as forty years before, a fascinating but incompletely solved problem "

—Wilhelm His, in 1933, at the age of seventy
(Klinische Wochenschrift)

WILHELM HIS, THE YOUNGER, was born in Basle, Switzerland, on December 29, 1863 His father at that time was professor of anatomy at the University of Basle Wilhelm His, the elder, was called to the University of Leipzig in 1872 There he shared with Carl Ludwig the chair of anatomy vacated by Weber Wilhelm Junior studied at the gymnasium in Leipzig Leipzig was the center of culture in Southern Germany and the young student's outlook was broadened by his many contacts with music and the arts Young His spent the last two years of his gymnasium at Basle He studied at the Universities of Leipzig, Strasbourg, Bern, and Geneva He was graduated from the University of Leipzig in 1888

In 1889 he became an assistant in the medical clinic at Leipzig under Heinrich Curschmann In this clinic Krehl was a first assistant and Ernst Romberg was a co-assistant In 1893 a small volume entitled "Arbeiten aus dem medizinischen Klinik zu Leipzig" was published from the clinic In addition to some fundamental studies by Krehl and Romberg there appeared in this work the first observations of His concerning the atrioventricular bundle of the heart and its function. We are presenting in translation His's summary, as recorded in this early paper Of the experimental work which led him to his discovery, His wrote, in 1933

"It was the current teaching of the time that the ganglia are the autonomic centers of the heart Only Engelmann, in Utrecht, and Gaskell, in Cambridge, held the belief, based on their experiments, that the heart muscle itself is able to originate rhythmic stimuli I was present one day when Krehl and Romberg discussed these subjects I proposed to them to study from an embryological point of view the development of the heart to try to ascertain whether or not the heart is able to beat before it has nerves and ganglia At that time I had finished an embryological paper under my father's direction and was, therefore, familiar with the technique necessary for such a study

"I followed the development of the cardio-nervous system through several vertebrates and could prove that in all these animals the heart beats before it receives cerebral spinal nerves or ganglia One point remained mysterious, namely, the conduction of the stimulus from one part of the heart to the other

"Gaskell had shown that in the frog and turtle the conduction is made by way of the muscles I tried to prove such a muscular connection in the adult mammal and in human beings by examining serial sections in the various embryonic stages I finally found these muscular connections and described them in 1893 Few have read this paper "

In His's original paper of 1893 he also described the first instance of the occurrence of Adams-Stokes syndrome in Germany, and he was able to demonstrate the pathologic seat of the interrupted conduction in the bundle

His, in 1891, became associate professor of internal medicine at the University of Leipzig In 1901 he went to Dresden and in 1902 he succeeded Friedrich Muller as director of the medical clinic at Basle At Basle, His became interested in physiologic chemistry and with his assistant, Bloch, he worked on the biochemic aspects of uric acid

In 1907, His succeeded Ernst Victor von Leyden in Berlin Here, he continued his studies on the heart and also gave much attention to the metabolism of gout and to diseases of the blood In Berlin he was popular as a teacher He also served for some time as editor of the "Zeitschrift fur klinische Medizin," and later became director of the first medical clinic at the University of Berlin

His had become a naturalized citizen of Saxony in 1895, so that at the outbreak of the World War he joined the German army as a voluntary soldier in spite of his being Swiss by birth The German surgeon-general, Lieutenant-General Otto von Schjerning, created a special appointment for him, that of consulting internist on September 2, 1914, and he was assigned to the theater of the war in East Prussia His observations on, and measures against, epidemics proved to be very valuable and von Schjerning sent him on missions to Turkey, Asia Minor, the Western theater of war, Russia and the Ukraine During the war, he was the first to describe (February 23, 1916, at Warsaw) Volhynia fever (trench fever), which he named after a district in Russia in which he observed the disease His's experiences in the war were set forth in a small book, "Die Front der Arzte" This was published in 1931, and proved so fascinating that it had wide circulation outside of the medical profession In 1933 the Association of Military Surgeons of the United States sponsored an English translation of the book by Dr Gustavus M Blech, of Chicago

The outstanding characteristic of His's personality was a lofty standard of education and culture He was a good violinist, a talented painter, and had a keen appreciation of art, literature, and history

His suffered from emphysema and died on October 10, 1934

THE FUNCTION OF THE EMBRYONIC HEART AND ITS SIGNIFICANCE IN THE INTERPRETATION OF THE HEART ACTION IN THE ADULT*

By

DR. WILHELM HIS, Jr.

After extensive investigation I was able to find a muscle bundle which connects the auricular and ventricular septal walls, and which apparently had not been observed before, because it is only visible in its entire distribution when the septal walls are cut exactly in the horizontal direction I was able to recognize the course of this bundle on such sections and on serial sections, and have proved its presence in an adult mouse, in a new-born dog, in two new-born infants and one adult, about 30 years of age The bundle arises from the posterior wall of the ventricle near the auricular septum in the atrioventricular groove, it joins the upper edge of the ventricular septum and ramifies, coursing on the septum anteriorly until it branches near the aorta into a right and a left branch, the latter terminating in the base of the aortic cusp of the mitral valve

I cannot state with certainty whether this bundle actually conducts the impulses from the auricle to the ventricle, as I did not perform any experiments dealing with the severing of the bundle Its presence, in all events, is contrary to the opinion of those, who, in the absence of such a muscular connection between the auricle and ventricle, attempt to prove the necessary presence of a nerve conduction

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*His Wilhelm, Jr Die Thätigkeit des embryonalen Herzens und deren Bedeutung für die Lehre von der Herzbewegung beim Erwachsenen, *Arbeiten aus der med Klin zu Leipzig*, 1893, pp 14-50 Translated by F A W

1896

FRANCIS H. WILLIAMS

FLUOROSCOPY OF THE HEART

FRANCIS HENRY WILLIAMS

(Courtesy American Journal of Roentgenology and Radium Therapy)

FRANCIS HENRY WILLIAMS

(1852-1936)

FRANCIS HENRY WILLIAMS, a pioneer in the development of roentgen-ray and radium therapy, was born in Uxbridge, Massachusetts, the son of Henry Willard and Elizabeth Dew Williams. He attended the Massachusetts Institute of Technology and was graduated from that institution in 1873. The following year, he traveled around the world. During this trip he attended a meeting in Japan as a member of the United States Transit of Venus Expedition.

Following his journey, Williams began the study of medicine at Harvard University, and in 1877 he received the degree of Doctor of Medicine from that institution. He spent the next two years in Europe in postgraduate study.

In 1879 he established himself in Boston and began the practice of medicine, interesting himself particularly in diphtheria. In Boston he spent his long and useful career. In 1884, he was appointed instructor in materia medica in the Harvard Medical School, and he later became assistant professor of therapeutics at that school.

In 1891 he was married to Anna Dunn Philips, of Boston.

Williams first began his work on the roentgen rays in 1896 (only a year after their discovery) while he was visiting physician to the Boston City Hospital. Because the Boston City Hospital at that time did not possess the necessary facilities, his first patients were examined at the Rogers Laboratory of Physics of the Massachusetts Institute of Technology. Eventually, the trustees of the Boston City Hospital granted him quarters in the basement and there he worked until 1915. In 1913, with the establishment of the roentgen-ray department at Boston City Hospital, he was appointed senior physician.

Williams was quick to observe the diagnostic value of the roentgen rays. We are reprinting his important article, which was one of the first published accounts of fluoroscopy of the heart (1896). Therein, in addition to showing how pathologic conditions may be observed by means of the fluoroscope, he suggested correlating roentgen-ray studies with data obtained by other means of physical diagnosis.

By 1898, through the medium of the fluoroscopic screen, Williams was able to demonstrate the application of the roentgen rays to the diagnosis of thoracic aneurysm, pericardial effusion, cardiac hypertrophy, cardiac transposition, emphysema, pleurisy with effusion, pneumothorax, including hydropneumothorax, and pulmonary tuberculosis.

In 1899 Williams and Walter B. Cannon, with the aid of the fluoroscope, demonstrated important physiologic facts concerning the stomach and intestines. In two children, they were able to show the relationship of visceral position to suggestive posture, the excursion of the stomach during respiration, and the changes in its shape during digestion.

In 1901, Williams published his comprehensive work, "Roentgen Rays in Medicine and Surgery." This was a successful publishing venture and a second edition appeared in 1903.

With his friend, Dr. William Rollins, Williams perfected new instruments to aid the practical use of roentgen rays and radium. Chief among these was the invention

of the fluorometer in 1902 This instrument is used for measuring the quantity of roentgen rays given out by the roentgen-ray tube It has also been used for the quantitative estimation of beta particles (electrons) and gamma rays given off by the radium (and radioactive) salts Williams and Rollins also improved on the fluoroscopic screen and invented a mechanical stereoscopic fluoroscope

Dr Williams was the recipient of many honors He was a fellow of the American Association for the Advancement of Science, and a fellow of the American Academy of Arts and Sciences He served as president in 1917-1918 of the Association of American Physicians He was an active member of the Massachusetts Medical Society, the American Medical Association and the Société de Radiologie médicale de France He was a corresponding member of the K K Gesellschaft der Arzte in Vienna, and an honorary member of the American Radium Society, the American Roentgen Ray Society, and the Radiological Society of North America

It is significant, as Percy Brown has pointed out, in ''American Martyrs to Science Through the Roentgen Rays,'' that Dr Williams realized the dangers inherent in roentgen rays almost from the beginning of his career Brown quotes Williams as saying [1] ''I thought that rays having such power of penetrating matter, as the x-rays had, must have some effect upon the system, and therefore I protected myself '' So far as is known, Williams never was afflicted by the terrible ulcerative and carcinomatous processes that attacked other pioneer workers who did not venture to protect themselves against the roentgen rays

Dr Williams retired from active practice in 1930, at the age of seventy-eight He continued to contribute to the literature of his field until his death in 1936 At the age of eighty-three he published a small volume entitled ''Radium Treatment of Skin Diseases, New Growths, Diseases of the Eye and Tonsils ''

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[1]Brown Percy *American Martyrs to Science Through the Roentgen Rays,* ed 1 Springfield 1936 Charles C Thomas p 17

A METHOD FOR MORE FULLY DETERMINING THE OUTLINE OF THE HEART BY MEANS OF THE FLUORESCOPE TOGETHER WITH OTHER USES OF THIS INSTRUMENT IN MEDICINE*

By

FRANCIS H. WILLIAMS, M D.

A SHORT account of some of my work on the applications of x-rays in medicine was read at the meeting of the Association of American Physicians held in April last and has been published in their "Transactions" I now wish to speak further of some of the uses of the fluorescope in medicine, leaving a fuller discussion of them and of my observations relating to physiology and diagnosis, to a later time, when I shall hope also to describe the methods of examination that I have employed

The picture which presents itself to the eye when the body is examined by the fluorescope is full of interest The trunk appears lighter above than below the diaphragm and the rise and fall of the muscle are distinctly seen, the chest is divided vertically by an ill-defined dark band which includes the backbone, and each side is crossed by the dark outline of the ribs, the spaces between which, are the brightest portion of the picture One also sees the pulsating heart, especially the ventricles, and under favorable conditions the right auricle and left auricle, but it is difficult to separate the latter from the pulmonary artery, a small portion of one side of the arch of the aorta may be seen in the first intercostal space to the left of the sternum The organs of the abdomen are much less readily observed, but the presence of a piece of lead or of substances impermeable to the Rontgen rays may be detected in them The neck and face may be reached with the fluorescope, and in the arms and legs the bones and certain foreign substances may be seen The head is the least promising field

In examining the heart by means of percussion, we can usually determine its left border, but we cannot find its lower border Now let us see what can be done in this direction by means of the x-rays The constant motion of the heart and diaphragm interferes with the use of radiography but renders fluorescopy all the more valuable The lungs and the organs adjacent to them are the parts of the body which best lend themselves to fluorescopic examination, because of the great difference in

*Boston M & S J 135 335-337, 1896

density between the former and the latter, or, in other words, of permeability to the x-rays The lungs being less dense than the neighboring organs allow the x-rays to pass through them more readily, and thus appear light against a darker back-ground formed by the heart and parts of the liver and spleen, which, owing to their density, are less permeable by the rays and thus appear dark when seen through the fluorescope, that is, there is contrast

The heart lies in such a position, however, that ordinarily but a certain portion of its outline may be seen with a fluorescope, a horizontal plane may be imagined through the body, when in a standing position, that would pass through the heart, liver and spleen, as these latter organs overlap the heart to some extent, but it is readily possible to isolate the heart, as it were, by the contraction of the diaphragm, the organs below the heart being then depressed, the overlapping is avoided and the heart being more closely surrounded by transparent lung tissue the whole of the apex, and part of its lower border come into view and may be drawn on the skin A suitable position of the Crookes tube of course facilitates this end somewhat It is desirable to see as much as possible of the heart at one time in order to best estimate its condition—then if necessary we may study one or another portion separately—and by means of this fluorescopic examination we can follow a larger portion of its outline and gain more information as to its size, position and action than has hitherto been within our reach I may add here that I have made an instrument that enables me to listen to the heart-sounds while watching the pulsating organ

The character of the revelations which are made to us by a fluorescopic examination of the heart may be most briefly suggested by Figs 1, 2, 3 and 4, taken from photographs of lines traced on the skin, which follow the outlines of the organs as seen through the fluorescope The patients from whom these photographs were taken were lying on a canvas stretcher and the Crookes tube was placed under and about a foot below the trunk These illustrations have been selected from a number of photographs I have thus far made

Case I Fifty-seven years old The Crookes tube was placed eighteen inches away from and under the point indicated by the black dot in Fig 1 The heavy crossed lines indicate the sixth rib, the full lines, what was seen in the fluorescope, the broken line, the border of the heart obtained by percussion, which on the left side coincides with the line as seen in the fluorescope The lower border of the heart cannot be got by percussion, and is seen in the fluorescope only during deep inspiration No apex beat was felt The full parallel lines on either side of the body mark the diaphragm in ordinary expiration and deep inspiration respectively

Case II Twenty-seven years old The full curved line on the left (see Fig 2), as far as the dot which marks the apex beat, and the broken line

inside, indicate both what was seen in the fluorescope, and what was obtained by percussion—the full line during ordinary expiration, the broken line during deep inspiration The continuation of the full curved line (beyond the broken line) that runs towards the sternum marks the lower

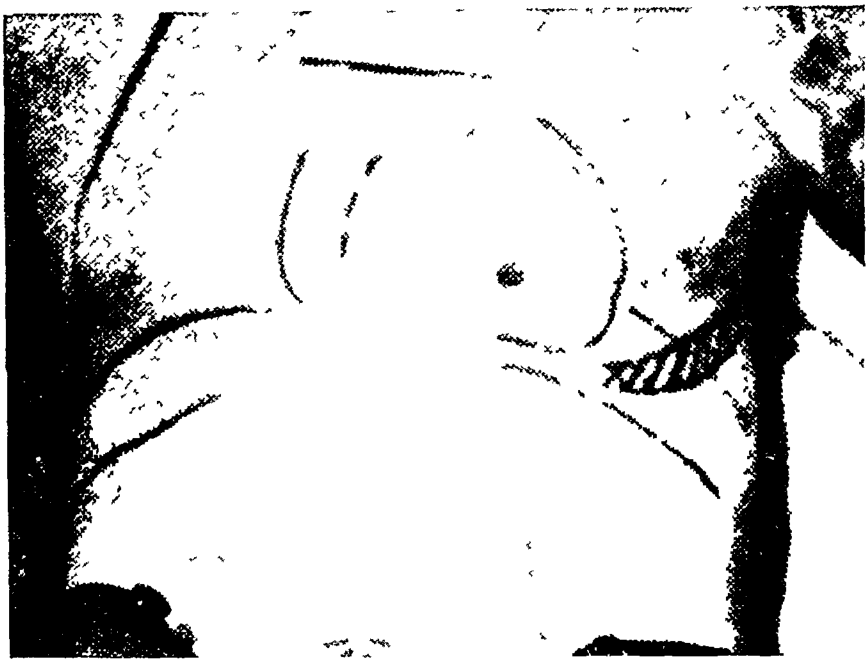

Fig 1

Fig 2

border of the heart, which as above stated, is obtained by the fluorescope, and this only during deep inspiration The two parallel lines on the left mark the diaphragm—the upper in ordinary expiration, the lower in deep inspiration The difference between these is greater than in Figs 1 and 3

Case III A boy, eleven years old The lines in Fig 3 indicate what was seen in the fluorescope The full curved line on the left marks the border of the heart as seen during ordinary expiration, the broken line inside, during deep inspiration, the two parallel lines on either side, the diaphragm in ordinary expiration and deep inspiration, the black dot, the point where the apex beat was felt

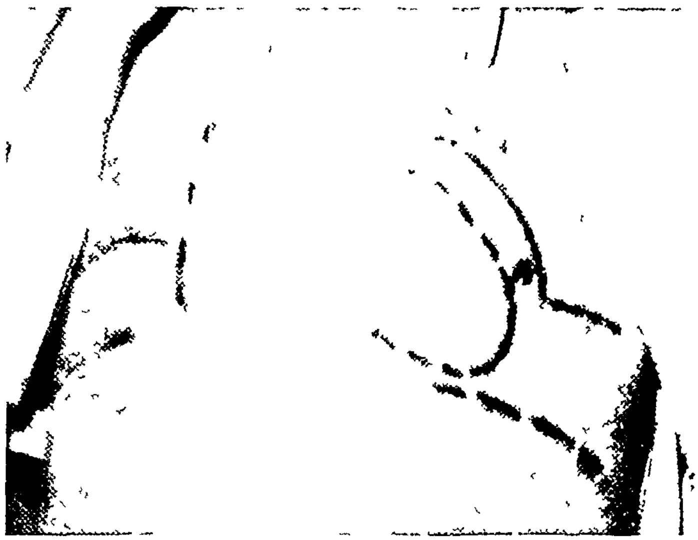

Fig 3

Fig 4

Case IV Fifty-eight years old Enlarged heart, examination with the fluorescope the left and part of the lower border of heart are seen (Fig 4) and the diaphragm lines already alluded to in the other cases The difference between these last mentioned lines is less than usual

These outlines of the heart are obtained with the fluorescope when the lungs are normal, when abnormal, on the contrary, as in tuberculosis, for instance, if the lungs are much involved, the outlines of the heart as well as those of the liver and spleen are obliterated as the lungs become impermeable to the x-rays, and therefore the outlines of these organs cannot be distinguished

In another abnormal condition of the lungs, namely, emphysema, I have observed that the heart, liver and spleen are still more distinctly visible than when the lungs are normal This is because in emphysema the volume of the lungs being increased, and yet retaining their transparency, the diaphragm is depressed and a part of the lungs intruding themselves between the sternum and heart, and their edges between the chest wall and the liver and spleen separate two dense tissues, thus rendering a larger portion of the outlines of the heart, liver and spleen visible in the fluorescope than when the lungs are normal This condition of things assists in making a diagnosis of emphysema of the lungs On the other hand, in this disease the outline of the heart is ill defined, or in some cases, impossible to define by percussion, therefore, the fluorescope in emphysema renders especial service

The fluorescope also reveals the presence of certain aneurisms, as they are dense, if their outline encroaches upon the visible portion of the comparatively transparent lung tissue, likewise, in connection with other physical signs, the presence of fluid in the pleural and pericardial sacs, the presence of pneumonia, edema of the lungs, infarctions, tuberculosis, hydro-thorax, pneumo-thorax, etc Further, it assists in diagnosis in excluding certain diseases by showing that the lungs are clear, this would be of importance in life insurance examinations

I have examined about forty cases of tuberculosis, and find not only that the fluorescope is of value in determining the extent of the disease, but also sometimes reveals its location, where and when it would otherwise have been unsuspected

In pleurisy with effusion the outline of the diaphragm is less defined or obliterated altogether according to the amount of fluid present, as are also some of the ribs in the upper portion of the affected side, the lung is also denser, being compressed by the fluid, if there is much effusion I observed in one case that the line separating the fluid surrounding the lower part of the lung from the compressed upper portion ran from about the junction of the sixth rib with the sternum towards the outer end of the clavicle, this line as seen in the fluorescope corresponded with the one found by percussion

All of the fluids withdrawn from the body which I have thus far examined with the fluorescope and by means of radiographs have rather less permeability to the rays than water About an equal thickness of the heart, liver, spleen, kidney, and muscle after death were about alike in permeability and were not much more of an obstacle to the rays than an equal bulk

of water. Thus it is easy to understand why fluid in the pleural cavity dulls or obliterates the outline of the adjacent organs, when the lungs become dense by disease they may obliterate not only the outlines of the ribs, but also those of the liver, spleen, and heart, as I have already indicated. When there is not marked contrast between the intercostal spaces and the ribs, or when the outlines of the clavicle and of adjacent organs are not defined, it should always arouse suspicion of something abnormal in the lungs or pleura.

The application of x-rays to surgery has hitherto formed the prominent side of their usefulness, I have pursued my investigations believing that it would be possible to demonstrate their usefulness in medicine, as distinguished from surgery, and am now confident that the advances these x-rays render possible in medical diagnosis are great, and that they will prove a more valuable instrument in the hands of the physician than of the surgeon. I have found them especially useful in diseases of the heart and lungs. We may now look where we have previously only been able to listen, and sometimes to hear but imperfectly. The advance consists not alone in what we can now see that we could not see before, but also in that we can by hand and ear, and eye together strengthen and confirm these separate observations beyond their respective limitations, singly they are beams, together an arch which justifies a heavier weight of inference.

Most of the work here described has been done in the x-ray room at the Boston City Hospital, the cordial interest of the Trustees and of my colleagues on duty has facilitated greatly the carrying out of these observations, and I shall always be indebted to Prof. Charles R. Cross of the Massachusetts Institute of Technology and to the assistants in his laboratory for the opportunity of studying the physics relating to the x-rays.

1897

SIR WILLIAM H. BROADBENT

ADHERENT PERICARDIUM

SIR WILLIAM HENRY BROADBENT

(Courtesy Charles C Thomas)

SIR WILLIAM HENRY BROADBENT

(1835-1907)

"I trust in God from day to day, seeking and asking for nothing beyond the happy mean, neither poverty nor riches, but grace to serve Him and do the work He has given me to do."

—Sir William Henry Broadbent,
quoted in *The Practitioner*

WILLIAM HENRY BROADBENT was born at Longwood, a small village in Yorkshire, England, on January 23, 1835. His father was a manufacturer of woolens and desired that his son should succeed him in business. Thus, after young Broadbent received some academic training at Huddersfield College, he left school at the age of fifteen to learn the fundamentals of his father's business. After a trial of two years, not finding the work to his liking, the future baronet embarked on a career in medicine, his choice of profession.

At the age of seventeen, Broadbent became an apprentice to a surgeon in Manchester. The apprenticeship was for a five-year period and the fee was to include the expenses of a medical course at Owens College. Broadbent was a brilliant student and in spite of his meager resources and the need for his daily visits to patients, dispensing medicines, and attending lectures at the Royal School of Medicine he won medals in botany, materia medica, anatomy, physiology, chemistry, midwifery, surgery, and operative surgery.

In 1856 Broadbent went to London for the first examination for the degree of Bachelor of Medicine at the University of London. There, too, he showed his intellectual superiority by winning gold medals in anatomy, physiology, and chemistry. In 1857 he passed the final examination of the Conjoint Board of the Royal Colleges of Physicians and Surgeons in London, and in 1858 he returned for his final examination for the degree of Bachelor of Medicine at the University of London. There he received the gold medal in obstetrics and came away with first-class honors in medicine.

As soon as he was qualified, he accepted the post of obstetric officer at St. Mary's Hospital in London. From this time until 1896 he was actively engaged in duties at St. Mary's. On the termination of his appointment as obstetric officer, Broadbent in 1859 became resident medical officer. The following year he was appointed pathologist to the hospital and also was chosen to lecture on physiology and zoology in the hospital's medical school. At a later date he became curator of the museum. In 1860, also, he received the degree of Doctor of Medicine from the University of London, and in the following year became a member of the Royal College of Physicians. In 1864, on the retirement of Dr. Chambers, Broadbent was appointed assistant physician.

At St. Mary's, Broadbent worked under Sibson, who at that time was a leading specialist on thoracic diseases. Sibson loved his specialty to such an extent that he was known to spend as long as half an hour listening to one murmur. Sibson was the source of Broadbent's inspiration in his subsequent work on diseases of the heart.

In 1871 Broadbent was promoted to be physician in charge of the in-patients at St Mary's He was also appointed lecturer in medicine, a position which he held for seventeen years Broadbent was connected with many other hospitals in addition to St Mary's He served as physician to the London Fever Hospital from 1860 to 1879, and afterward acted as consultant He was also on the medical staffs at Western General Dispensary and the New Hospital for Women

Broadbent's output of original papers was large These papers were later collected by his son, Dr Walter Broadbent, and published in book form[1] Although Broadbent was naturally interested in the whole field of internal medicine, he spent considerable time on the study of the nervous system and also the cardiovascular system

An article that contributed to his early pre-eminence in the field of neurology was his paper on sensory-motor ganglia,[2] in which Broadbent explained the immunity to paralysis of bilaterally associated muscles in hemiplegia The subject of aphasia was also of interest to Broadbent and he contributed several interesting articles on the condition Broadbent also wrote on chorea, syphilitic affections of the nervous system, ingravescent apoplexy and alcoholic spinal paralysis

Broadbent's great interest in cardiology is reflected in his lectures on "Prognosis in Valvular Disease of the Heart" He delivered these before the Harveian Society in 1884 In 1887 he delivered the Croonian lectures on the pulse before the Royal College of Physicians in London These he elaborated upon in his book, "The Pulse," published in 1890 by Carsell and Company, London This classic book shows what was accomplished with the use of the finger before the days of clinical employment of the sphygmometer

In 1891, when Broadbent was invited to give the Lumleian lectures, he chose for his subject, "Prognosis in Structural Diseases of the Heart" These lectures as well as his Harveian lectures were the basis for his book on heart disease published in collaboration with his elder son in 1897 Included therein was Broadbent's first description of the signs of adherent pericarditis This we are reprinting According to his own account (published in 1898) Broadbent established this famous cardiac sign as early as 1868,[3] but it was not until 1895 when his son, Walter, made note of it in the "Lancet"[4] that it received its first publicity

In 1892 Broadbent was appointed physician in ordinary to the Prince of Wales, who was later to become King Edward VII The following year Queen Victoria conferred a baronetcy upon him on the occasion of the marriage of the Prince of Wales, whom Broadbent had attended two years previously for an attack of typhoid fever Sir William was later named physician in ordinary to King Edward VII, and in 1901 he was created a Knight Commander of the Victorian Order

Sir William was the recipient of many honorary academic degrees in recognition of his achievements He was awarded the degree of Doctor of Laws by Edinburgh University in 1898, and by St Andrew's University in 1899 In 1904 the University of Leeds made him an honorary Doctor of Science, and the University of Toronto granted him the degree of Doctor of Laws on the occasion of the meeting of the British Medical Association in the city of Toronto, in September, 1906

Broadbent was one of the organizers and was also the first president of the Entente Cordiale Medicale (1904) and at the banquet given in Paris in honor of

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[1]Broadbent, Walter *Selections from the writings, medical and neurological, of Sir William Broadbent,* London 1908 Frowde Hodder & Stoughton 444 pp

[2]Broadbent W H An attempt to remove the difficulties attending the application of Dr Carpenter's theory of the function of the sensori-motor ganglia to the common form of hemiplegia Brit & For M Rev 37 468-481 1866

[3]Broadbent, Sir W Adherent pericardium Tr M Soc London 21 109-122 1898

[4]Broadbent, Walter An unpublished physical sign Lancet 2 200-201 1895

English physicians he was invested with the Grand Cross and insignia of a Commander of the Legion of Honor of the Republic of France

Apparently Sir William enjoyed good health most of his life In 1906, however, he suffered a severe attack of pneumonia This was followed by empyema, which was successfully relieved by operation, but from which he did not fully recover He died on July 10, 1907

Sir William Broadbent in 1863 had married Eliza Harpin, by whom he had two sons and three daughters His elder son, Dr John Francis Broadbent, succeeded him in the baronetcy The younger son was Dr Walter Broadbent, to whom we have also alluded

CHAPTER XVII

DISEASES OF THE PERICARDIUM*

ADHERENT PERICARDIUM

By the term "adherent pericardium" is implied the existence of adhesions between the visceral and parietal layers of the pericardium, the result of pericarditis. They may be limited to fibrous bands stretching across the pericardial cavity, or they may be universal, in which case the pericardium and heart are so intimately connected that the pericardial cavity is entirely obliterated. Adhesions may also exist between the chest-wall or pleura and the pericardium, as a result of so-called mediastino-pericarditis. The adhesions if of old standing are tough and fibrous, so that the pericardium cannot be stripped from the heart without tearing the heart-substance. There is also commonly some fibroid change in the heart-wall due to substitution of fibrous tissue for muscle fibres damaged by previous inflammation. In the case of recent adhesions or lymph undergoing organization into fibrous tissue, the two layers of pericardium on being separated will present a honeycomb or bread-and-butter-like appearance, owing to the layer of thick, sticky lymph which coats the surface.

Physical Signs

The physical signs differ according as the adhesions exist only between the two layers of the pericardium, or between the pericardium and chest-wall, or adjoining pleura as well. In the latter case they are more numerous and distinctive. Among them are the following:

Fixation of the apex beat, so that it does not alter its position in deep inspiration and expiration or in change of posture of the body.

Systolic depression of one or more intercostal spaces to the left of the sternum, or of the lower end of the sternum and the adjoining costal cartilages, which may be caused by the heart dragging on them at each systole, through the agency of the pericardial adhesions. The systolic recession of spaces alone is, however, not a trustworthy indication, as it may be due to atmospheric pressure, more especially when the heart is much hypertrophied. When the costal cartilages or lower end of the sternum are dragged in there can be little doubt as to the diagnosis, as this could not be effected by atmospheric pressure.

*Broadbent Sir William H and Broadbent John F H *Heart disease with special reference to prognosis and treatment,* New York 1897 William Wood & Co

Systolic recession of the site of the apex beat is an important sign when a definite apex beat can be felt; when there is no palpable apex beat, systolic pitting over its site may be due to atmospheric pressure.

A diastolic shock may sometimes be felt on palpation with the flat of the hand over areas on the chest-wall where systolic recession is present. It is due to the elastic recoil of the chest-wall at the commencement of diastole as soon as the pulling force exerted during the systole ceases.

Systolic retraction of the lower portions of the posterior or lateral walls of the thorax may indicate the presence of a universally adherent pericardium. Such retraction may, however, be seen though the pericardium is not adherent to the heart, but only to a larger extent than normal to the central tendon of the diaphragm and the muscular substance on either side, and to the chest-wall as well. In such cases the heart is usually greatly enlarged and hypertrophied from old valvular disease. The explanation seems to be that the portion of the diaphragm to which the pericardium is adherent is dragged upwards at each systole of the heart, so that the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inwards and retracted.

The **descent of the diaphragm in inspiration** may be interfered with by pericardial adhesions between the heart and diaphragm, more especially if the pericardium is adherent to the chest-wall in front as well. This will be shown by impaired movement in respiration of the upper part of the abdominal wall in the epigastrium and left subcostal region.

The **area of cardiac dulness** will be increased, and will remain unchanged in inspiration and expiration, where there are extensive adhesions between the pericardium and chest-wall, as the lung, which normally overlaps part of the heart, will have been pushed aside, or perhaps have become involved in the adhesions, and be collapsed.

Enlargement of Heart.—It is common with adherent pericardium to find the heart, more especially the right ventricle, considerably enlarged, in the absence of valvular disease or other obvious cause to account for it.

It seems probable that such enlargement may be indirectly due to pericardial adhesions as follows. The heart becomes dilated during an attack of pericarditis, and, before it recovers its tone or can contract down again to its normal size, the pericardium becomes adherent and fixes it in this condition of dilatation, the right ventricle suffering more than the left, owing to its thinner walls, as well as for other reasons.

Hypertrophy and dilatation of the heart, more especially of the right ventricle, may therefore, in the absence of other obvious causes, such as valvular disease, high arterial tension, etc., to explain it, be a physical sign of considerable importance.

Diastolic collapse of cervical veins was held by Friedreich to be of great diagnostic value when accompanied by systolic retraction of spaces, but I have never found it to be of service.

Systolic emptying of veins on the surface of the thorax may sometimes be observed, due to suction action, induced by the walls of the internal mammary veins being dragged apart by pericardial adhesions during systole of the heart.

When there are no adhesions between the pericardium and chest-wall the physical signs that may be present will be limited in number. There will be no recession of spaces except as the result of atmospheric pressure, no fixation of apex beat, no diastolic shock. As the pericardium is normally attached by fibrous bands to the central tendon of the diaphragm and to the muscular substance on either side of it, there may be some interference with the movements of the diaphragm in respiration. There may also be cardiac enlargement indirectly due to the adhesions, but in such cases a diagnosis will usually have to be made from other indications than physical signs alone.

Symptoms.—The symptoms in themselves are not in any sense characteristic. They are usually such as arise from cardiac embarrassment, more especially from the giving way of the right ventricle, such as oedema of the extremities, enlargment of liver, ascites, dyspnoea, etc.

Diagnosis

The physical signs or symptoms of adherent pericardium, few of which may be present, are often in themselves insufficient to allow of a diagnosis being made, or even to arouse suspicion of its presence, but valuable help may be derived from careful consideration of the physical signs and symptoms together, and by balancing the former against the latter, so that the question is raised, "Do the physical signs present afford evidence of sufficient disease to account for the symptoms that have arisen?" When the symptoms are those of right ventricle failure, and are more severe than the physical signs present would lead one to expect, and have not been induced by undue exertion or imprudence, adherent pericardium must be thought of as being possibly responsible. For it is the right side of the heart more especially that is seriously hampered by pericardial adhesions, so that their presence may account for the unexpected breakdown of the right ventricle when the physical signs indicate that the valvular lesion is slight. It must also be borne in mind that the heart-wall has in all probability been weakened by the substitution of fibrous tissue for muscle fibres destroyed by inflammation at the time of the attack of pericarditis.

When with symptoms of right ventricle failure there is an absence of cyanosis, or of pulmonary congestion or lung mischief, this is further evidence in favour of adherent pericardium as a possible cause of the breakdown of the right ventricle. If by these means a suspicion of the presence of adherent pericardium has been aroused, confirmatory physical signs should be carefully sought for.

The above remarks apply to the question of diagnosis in cases where, with or without valvular disease, there is no history of pericarditis, and the adhesions are of old standing

In cases of pericarditis, which can be kept under observation after the attack, there will be less difficulty in arriving at a diagnosis, and the indications which would lead one to suspect that the pericardium was becoming adherent are as follows —

1 Prolongation of the attack of pericarditis evidenced by a harsh friction rub over the praecordial area, which may persist for some weeks When at the margins of the area of cardiac dulness a pleuro-pericardial friction is also heard, it will indicate that adhesions are probably taking place between the pericardium and adjoining pleura or chest-wall as well

2 Permanent enlargement of the area of cardiac dulness to a marked extent after the subsidence of the pericarditis

3 The occurrence of symptoms of right ventricle failure after a period of temporary improvement, there being no apparent exciting cause for the breakdown of the right ventricle Damage to the cardiac muscle by fresh myocarditis may, however, be responsible, and should be first excluded

Prognosis

When the heart remains normal in size, and there are no adhesions between the pericardium and chest-wall, the universal adherence of the pericardium to the heart may not in an adult tend to materially shorten life When the heart is enlarged, or when the pericardium is also adherent to the chest-wall, the prognosis is more serious When adherent pericardium exists as a complication of valvular disease, it is still more likely to prove fatal eventually, by so hampering the right ventricle as to prevent its recovery when once compensation has broken down The detection of adherent pericardium has also an important bearing on prognosis, inasmuch as it affords presumptive evidence of fibroid change in the heart-wall, and therefore renders the outlook even more unfavourable

Treatment

The discovery of adherent pericardium, when present, is important from the point of view of treatment, not because anything can be done to remedy or remove the pericardial adhesions, once they are formed, but because, when it is present, it will be necessary to impose additional restrictions on the patient, so that no undue risks may be run of upsetting the compensatory balance, which would only be restored with great difficulty

1903

WILLEM EINTHOVEN

THE ELECTROCARDIOGRAPH

WILLEM EINTHOVEN

(Courtesy Heart)

WILLEM EINTHOVEN

(1860 1927)

"Honours, however, were to him a smaller recompense than was the knowledge of the benefits which his long and arduous work had con ferred upon his fellow men "

—Sir Thomas Lewis, on
Willem Einthoven *(Heart)*

ONE OF the greatest advancements in modern cardiology was the conception of a means to study the electrical changes which accompany each heart beat The development of electrocardiography and with it modern cardiology owes to Einthoven a great debt

Willem Einthoven was born in May, 1860, in the Dutch East Indies (Semarang, Java), but spent most of his life in Holland There he studied at the University of Utrecht from 1879 until 1885, in which year he received his Doctor's degree at the age of twenty-five

Even as a medical student he had showed a remarkable interest in the physical sciences, but not until 1886, when his paper, "On the Law of Specific Nerve Energies," was published, did his reputation in this field become established

In 1886 Einthoven also published his inaugural thesis, "On the Influence of Color Differences in the Production of Stereoscopic Effects " Einthoven's basic interest in the physical sciences is well reflected in this paper In 1886, moreover, he was called to the chair of physiology and histology at the University of Leyden, in which post he was active for forty years until his death

Einthoven contributed much to the understanding of the physiology of the bronchial musculature and to the knowledge of the physiology of the eye, but his greatest gift to medicine was his discovery of the string galvanometer This pioneer contribution elucidated the physical principles of the electrocardiogram and enabled cardiologists to study, accurately, the biologic and physiologic activities of the heart

At the end of the Eighteenth Century, Luigi Galvani (1737-1798), professor of anatomy at the University of Bologna, became interested in animal electricity By accident one day he recorded a remarkable observation A dissected frog had been placed on a laboratory table near an electric machine Galvani's assistant had touched the nerves of the frog's leg with a knife, and the leg contracted vigorously Galvani, intensely curious, investigated the phenomenon, finally discovering that the leg would contract in such a manner only when the electric machine was sparking Leaman quoted Galvani of writing that "strong contraction took place in every muscle of the limb and at the very moment when the sparks appeared "

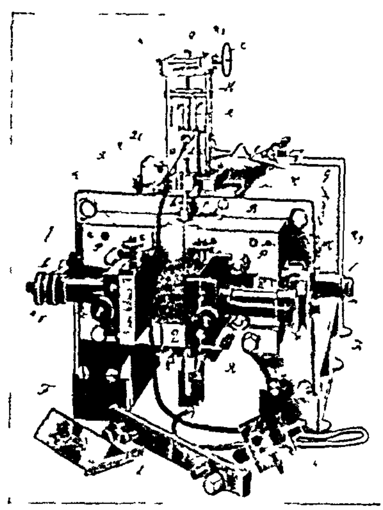
In many different experiments, Galvani later studied contractions of the muscles by using different methods of electrical stimulation, including lightning

In 1842 Carlo Matteucci (1811-1868), another Italian, advanced the knowledge of electrical conduction In an experiment he placed the cut, central end of the sciatic nerve of one leg of a frog on the muscles of the opposite leg When he stimulated the

sciatic nerve on the intact side, both muscles contracted, although only the normal side had been stimulated This observation was important in advancing knowledge of the influence of the electric current on nerves and muscles

In 1856 Albert von Kolliker and Muller discovered that an exposed frog's heart produced an electric current which accompanied each beat In 1878 John B Sanderson and F J M Page in England were finally able to record by means of the capillary electrometer, for the first time, the current produced by the action of the heart

The next advance in the development of electrocardiography was contributed by Augustus D Waller, who published a paper in 1887 entitled "A Demonstration on Man of the Electromotive Changes Accompanying the Heart's Beat"[1] This paper we reprint on pp 656-661 Waller showed that the currents of the heart could be studied without opening the thorax of laboratory animals, that these studies could be made by connecting the surface of the body of such animals with electrodes to which the capillary electrometer was attached These connections afterward were known as leads Waller also demonstrated that the current of the heart in human beings could be studied in the same way



Einthoven's string galvanometer

(Courtesy Ciba Symposia)

Much difficulty, however, was encountered with the capillary electrometer This instrument consisted of a column of mercury contained in a vertical glass tube, one end of which was dipped into sulfuric acid The mercury in this instrument would be disturbed by the electrical charge passing through it and the up and down movements of the mercury were photographed on a moving sensitized plate Since the mercury, because of its inertia, produced curves which were not exact, the resulting recordings were not accurate measurements of the electrical changes accompanying the heart beats

Einthoven, in 1903, employed the galvanometer, the invention of Johannes S C Schweigger (1779-1857), of the University of Halle, to measure the electric current

[1] J Physiol 8 229-234 1887

produced by the action of the heart. By the use of his string galvanometer, Einthoven perfected Schweigger's invention, which meant that he introduced a practical method for electrocardiography

Einthoven described the physical principles of the electrocardiogram and made pioneer contributions to the physiologic uses in which it may be utilized

Soon Sir Thomas Lewis began to use the instrument in England and to correlate clinical data with observations arising from use of the new instrument, and the electrocardiogram thus became an indispensable aid of modern cardiologists

In 1924, Willem Einthoven was awarded the Nobel Prize in medicine for his contributions which so greatly advanced the scientific study of modern cardiology and which are as important, perhaps, to the cardiologist as the use of digitalis is to the cardiac patient

THE GALVANOMETRIC REGISTRATION OF THE HUMAN ELECTROCARDIOGRAM, LIKEWISE A REVIEW OF THE USE OF THE CAPILLARY-ELECTROMETER IN PHYSIOLOGY[1] *

By

W. EINTHOVEN

Physiologic Laboratory in Leyden

UP TO the present time, the human electrocardiogram discovered by Aug D Waller[2] could be recorded only by means of the capillary-electrometer Simple inspection of the curve inscribed by means of this instrument results in an entirely fallacious representation of the variations of potential, which, as a matter of fact, actually existed If one desires accurate values of the latter, the form of the registered curve must be corrected for the size of the capillary tube used, the degree of magnification, and the speed of the photosensitive plate By this method one arrives at the construction of a new curve, the outline of which actually represents the variations of potential

In explanation of this fact, the following example will be offered [3]

Fig 1 represents the registered curve of Mr v d W by leads from the right and left hand, while Fig 2 represents the corrected curve

The differences are obvious One immediately likens the deflections C and D in the registered curve to the corresponding deflections R and T in the constructed curve Only the latter portrays an accurate comparison of the height of the deflections

What holds true for the electrocardiogram, also, in general, holds true for any other curve obtained by the capillary-electrometer, if one wishes to reproduce different changes in potential as occurring rather rapidly One is obliged in both instances, regardless of the method[4] employed, to

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*Einthoven W Die galvanometrische Registrirung des menschlichen Elektrokardiogramm zugleich eine Beurtheilung der Anwendung des Capillar-Elektrometers in der Physiologie, Pflüger s Arch f d ges Physiol 99 472-480 1903 Translation by F A W

[1]An investigation sponsored by the International Committee for the Unification of Physiologic Methods

[2]Phil Trans Roy Soc London, vol 180 p 169-194, 1889

[3]Pflüger s Arch Bd 605 101, 1895

[4]As shown on the preceding pages the practical methods of electrocardiography result in curves which may easily be analyzed in a graduated rectangular
see W Einthoven, Lippmann s Capillar-Elektrometer zur Messung
Potentialunterschiede Pflüger's Archiv, Bd 56 S 528 1894—Id
eschwindigkeit der Quecksilberbewegung in Lippmann s
rchiv Bd 60 S 91 1895—Id, Ueber die Form des
Pflüger s Archiv Bd 91 S 101 —Even earlier other capillary-electrometer curves were analyzed showing circular zero lines, that were taken on a sensitive plate fastened to a pendulum See G J Burch On a method of determining the value of rapid variations of a difference of potential by means of the capillary electrometer Proceed of the Royal Soc of London vol 48, p 89, 1890—Id On the time relations of the excursions of the capillary electrometer Philos Trans of the Royal Soc of London vol 183 A p 81 1892—J Burdon-Sanderson, The electrical response to stimulation of muscle Part II The Journal of Physiol vol 23 p 325, 1898—Eine Vereinfachung der Ausmessung in rechtwinkligen Coordinatensystem findet man bei S Garten, Ueber ein einfaches Verfahren zur Ausmessung der Capillarelektrometer-Curven Pflüger s Archiv Bd 89, S 613 1902 In the last paper a review of the other collections of measurements is given

construct a new curve from the one originally inscribed, whereby accurate measurements can be determined, which are chiefly concerned with different calculations

I have sought a method in which, as far as possible, the construction of a new curve could be avoided, and finally, to offer an instrument which primarily would satisfy the requirements of inscribing the electrocardiogram of human beings in approximately, at least, its correct proportions

This instrument—the string galvanometer—is essentially composed of a thin silver-coated quartz filament, which is stretched like a string, in a strong magnetic field When an electric current is conducted through this quartz filament, the filament reveals a movement which can be observed and photographed by means of considerable magnification, this movement is similar to the movements of the mercury contained in the capillary-electrometer It is possible to regulate the sensitivity of the galvanometer very accurately within broad limits by tightening or loosening the string

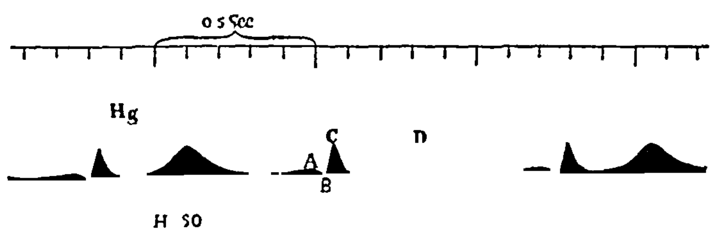


Fig 1

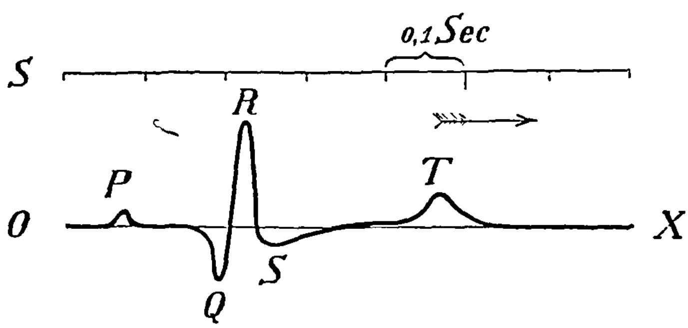


Fig 2

The theory, as well as the practical details, of this new instrument may be omitted at this time[5] It is not only of interest to discuss the similarity of the capillary-electrometer to this new instrument but also certain characteristics by means of which the string galvanometer distinguishes itself

It obviously is necessary to appreciate the fact that the movement of the string galvanometer measures electrical current, while the movement of the capillary-electrometer measures electrical potential Here I will state that each time that variations in potential or strength of current

[5]See W Einthoven, Ein neues Galvanometer Drude's Annalen der Physik Also in Onderzoekkingen Physiol Laborat Leyden 2 Reihe, Bd 5

occur, the mercury meniscus, as well as the filament, is in motion And during the phase of activity the capillary-electrometer must become alternately charged and discharged, while the string in the magnetic field develops an opposite electromotive force

In addition, under conditions of constant and great resistance, together with negligible induction, the strength of the current must at any moment be proportional to the effective electromotive force, such as is usually the case in electrophysiology Therefore, the differentiating principles of the electrometer and the galvanometer do not prevent the comparison of the two instruments

The string galvanometer, as compared with the capillary-electrometer, offers various advantages

1 In the first place, there are many instances, but particularly in the registration of the electrocardiogram of human beings, in which the deflections occur with more perfect aperiodicity, greater rapidity and with greater amplitude than those occurring with the capillary-electrometer The greater sensitivity of the string galvanometer must be ascribed partly to the fact that the finer quartz filament, in spite of its greater length, is nevertheless considerably lighter than the mercury column of the capillary-electrometer A quartz filament can be 10 times thinner than a mercury column, thus it is possible to have one with a cross section 100 times smaller, because the specific gravity of molten quartz is approximately 5 to 6 times less than that of mercury From this, one must calculate the active force of the moving quartz filament and take into consideration the fact that only the mid-portion, when observed microscopically, shows the greatest displacement All other portions of the filament have less movement

2 When one alters the tension of the filament, it becomes possible easily and accurately to regulate the sensitivity of the string galvanometer, and I am led to believe that the production of these curves satisfy the requirements of the International Committee for the Unification of Physiologic Methods It requires little effort to understand that a certain number of millimeters of a deflection is comparable to the number of millivolts of a deflection of a corresponding degree of tension or to the number of micro-amperes of current

3 With the string galvanometer, the deflection is exactly proportional to the strength of the current and maintains a complete equality between the deflections to the right and to the left, while with the capillary-electrometer, the proportionality between the deflections and the potential differences as it occurs with mercury and sulphuric acid, leaves much to be desired

4 With the capillary-electrometer, the excursions of the meniscus become restricted by the friction of the mercury and the sulphuric acid owing to their passage through a small tube Tiny invisible particles of dust may appreciably retard the movements of the mercury meniscus Many capillary tubes must be replaced after relatively short usage, owing to the fact

that the movement of the meniscus ceases. Electro-magnetic suppression (*Dampfung*) by chance can be influenced by variations in the strength of the field and conditions of the galvanometer.

5. The electrical isolation of the string galvanometer is more readily effected than is that of the capillary-electrometer, and a recession phenomenon does not occur in the galvanometer.

6. The filament offers certain advantages for observation in the projection of a clear-cut, magnified image. The aperture of the lens system, whereby the image is produced, is, as you know, of great importance in bringing about the necessary magnification. We can be certain that the use of a large aperture would be possible in the projection of the mercury meniscus of the capillary-electrometer, but it is evident that this offers no advantages as long as the illuminating lenses possess a smaller aperture. The illuminating lens in the capillary-electrometer is separated by the tubes or the chamber of sulphuric acid from the mercury, the focal distance of this lens thus must be relatively greater, its aperture small.[6] On the contrary, with the string-galvanometer, the distance from the filament can be shortened at will by manipulating the illuminating lens as well as the projection lens.[7] It is thus possible, with the dry system, to use a much larger aperture to good advantage.

One generally projects the image through a nearby effective cylindrical lens which concentrates the light on a photographic plate, perpendicularly to its axis. By this concentration of the light, the filament has a distinct advantage over the mercury meniscus, for, owing to the straight image of the filament, a sharp contrast between light and dark is maintained, while the curved mercury meniscus results in a less distinct outline.

7. And lastly, the operation of the string galvanometer is simpler. No special handling of the instrument is required when it is not in use. It is, thus, always ready for further use, which cannot be said of the capillary-electrometer.

On Plate VII the electrocardiograms of six persons are reproduced as registered by the string galvanometer. During the photography of the curves, the shadows are regularly projected in a co-ordinated manner on the sensitive plate, according to the accepted method of Garten.[8] The distance between the lines is arbitrarily chosen so that the entire field is divided into squares of about 1 sq. mm.

The speed of movement of the photographic plate conforms to 25 mm. per second, so that an abscissa of 1 mm. has a value of 0.04 second, while the tension of the filament is so adjusted, that an ordinate of 1 mm. corresponds to 10^{-4} volt of electromotive force. By the selection of these arbitrary values, the curves, nevertheless, meet the requirements of the International Committee.

[6] One observes that under these circumstances a more opaque image is dealt with.

[7] At least, if one does not wish to sacrifice other important advantages for projection.

[8] Dr. Siegfried Garten: Ueber rhythmische elektrische Vorgange im quergestreiften Skelett-muskel. Abhandl. der Kgl. sachs. Gesellsch. der Wissenshaften zu Leipzig, math.-phys. Classe, Bd. 26, Nr. 5, S. 331, 1901.

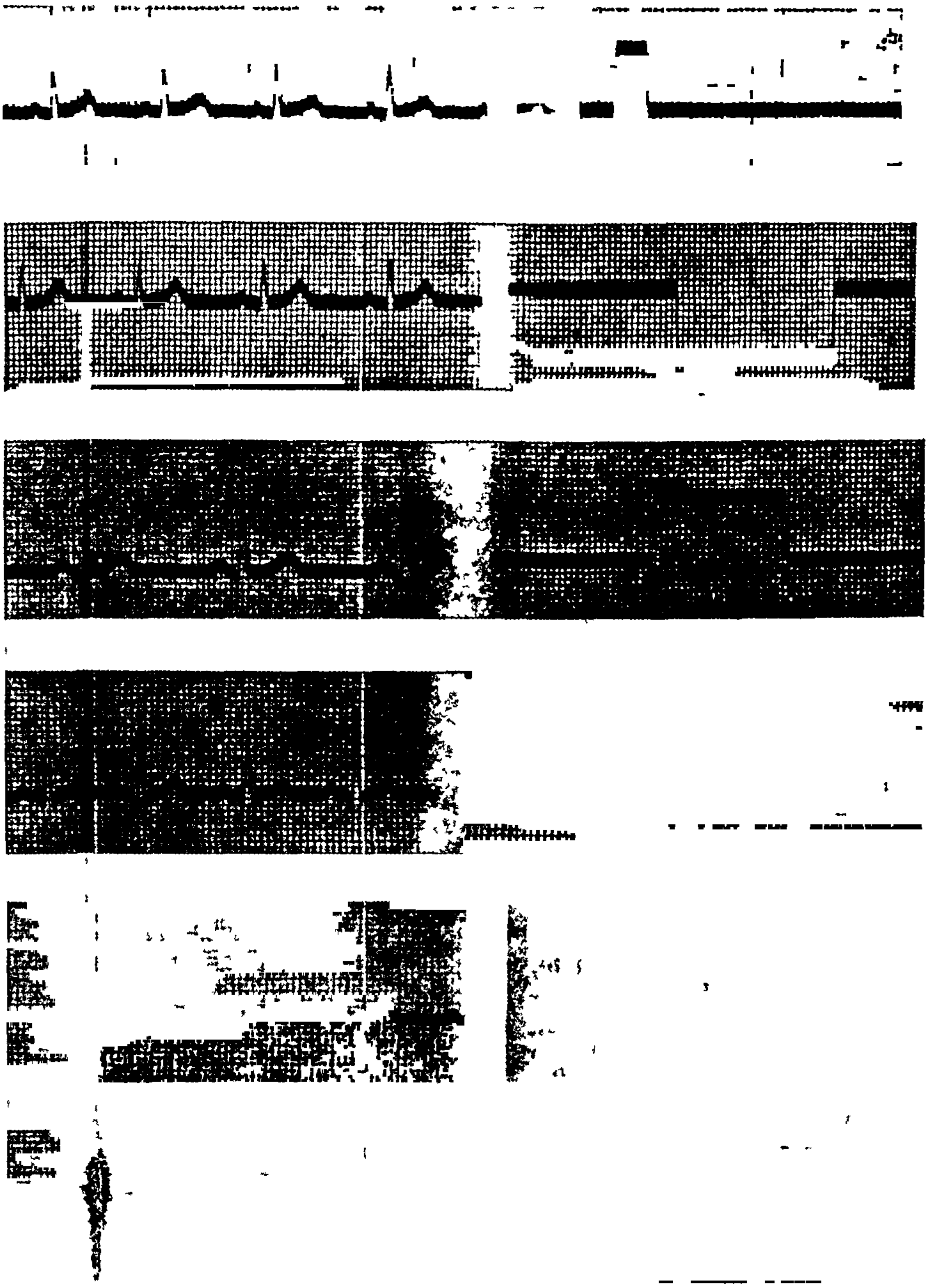


PLATE VII

Abscis 1 mm = 0 04 sec *Ordin* 1 mm = 10^{-4} volt

At the outer end of most of the photograms, a normal curve is pictured, which is presented to show that the resistance of the human body is compensated for by the introduction of 1 millivolt of electromotive force into the circuit. In Nr. 6 the inscription of the normal curve is omitted, while in Nr. 4, twice 1 millivolt is utilized. This figure shows the perfect proportionality that exists between the deflections and the electromotive forces.

Although the magnification is 660 fold, it must not be expected that the edges of the filament will be sharply defined; nevertheless, one is able with certainty to detect a 0.1 mm. displacement of the edge of the filament. For that purpose if we only consider one of the normal curves, i. e., Nr. 4, one can be readily convinced that a deflection of 1 and 2 millivolts, an accuracy as close as 0.1, amounts to 10 and 20 mm.

The movement of the quartz filament is aperiodic and rapid, as can be observed in the normal curves, so that the electrocardiogram as recorded by the string galvanometer accurately expresses the variations of potential existing at the time of registration between the right and the left hand of the person. This usually holds true, without appreciable error, for the lower peaks, P, Q, S, and T. A correction must be made for the high and pointed peak, R, especially in photograms Nrs. 3 and 4, whereby the upstroke of the peak is moved a trifle to the left. However, the correction required still remains slight, and its value for the displacement to the left is approximately less than 0.2 mm.

If one desires greater accuracy, the curves of the string galvanometer may be treated in the same manner as those of the capillary-electrometer, whereby values are derived from the registered curve for the construction of a new curve. But this in many instances will be unnecessary.

The photogram Nr. 3 represents the electrocardiogram of the same person whose capillary-electrometer curve is pictured in the text figure. If one compares the registered curve Nr. 3 of the plate with the earlier constructed curve in Fig. 2 of the text, it becomes obvious that both are very similar. The peaks, P, Q, R, S, and T, not only are present in both curves, but both curves have relatively similar excursions. In the constructed curve, the distance between the ordinates represents 1 millivolt and that between the abscissae, 0.1 second, while in the galvanometric curve, the distance between the ordinates corresponds to 1 millivolt and the distance between the abscissae, to 0.04 second. The galvanometric curve is consequently concentrated in the direction of the abscissae, a fact which becomes evident by casual examination of the curve.

The galvanometric curve, furthermore, gives the impression of symmetrical alterations from one peak to another, because the slight differences coincide better in the natural than in the constructed curve. According to the nature of the thing, only a limited number of points of the latter can be computed accurately, while the remaining portion of the curve must be constructed in such a manner as to connect, as nearly as possible, the points. However, the small differences are unimportant.

It is particularly gratifying that perfect confirmation of the results is found with the help of the newer instrument in various ways and by simpler means than those which were suggested previously with the aid of the capillary-electrometer, and through the medium of more or less complete calculations and construction. Then, by these means, a twofold proof is obtained: first, for the validity of the theory and the practical employment of the previously accepted method, and second, for the correctness and accuracy of the new instrument itself.

The six electrocardiograms in Plate VII were selected from a larger number and arranged according to the amplitude of the oppositely directed wave S (see the text figure). In 1 and 2, the curve adheres to the base line where S should appear along the iso-electric line of diastole; in 3 and 4, the S is shallow, and in 5 and 6, contrastingly deep. Nrs. 1 and 6 represent the extremes in our collection of available electrocardiograms, while the contrasting Nr. 8, the electrocardiogram of Mr. v. d. W., establishes a form of normal, to which all other numbers may be readily compared.

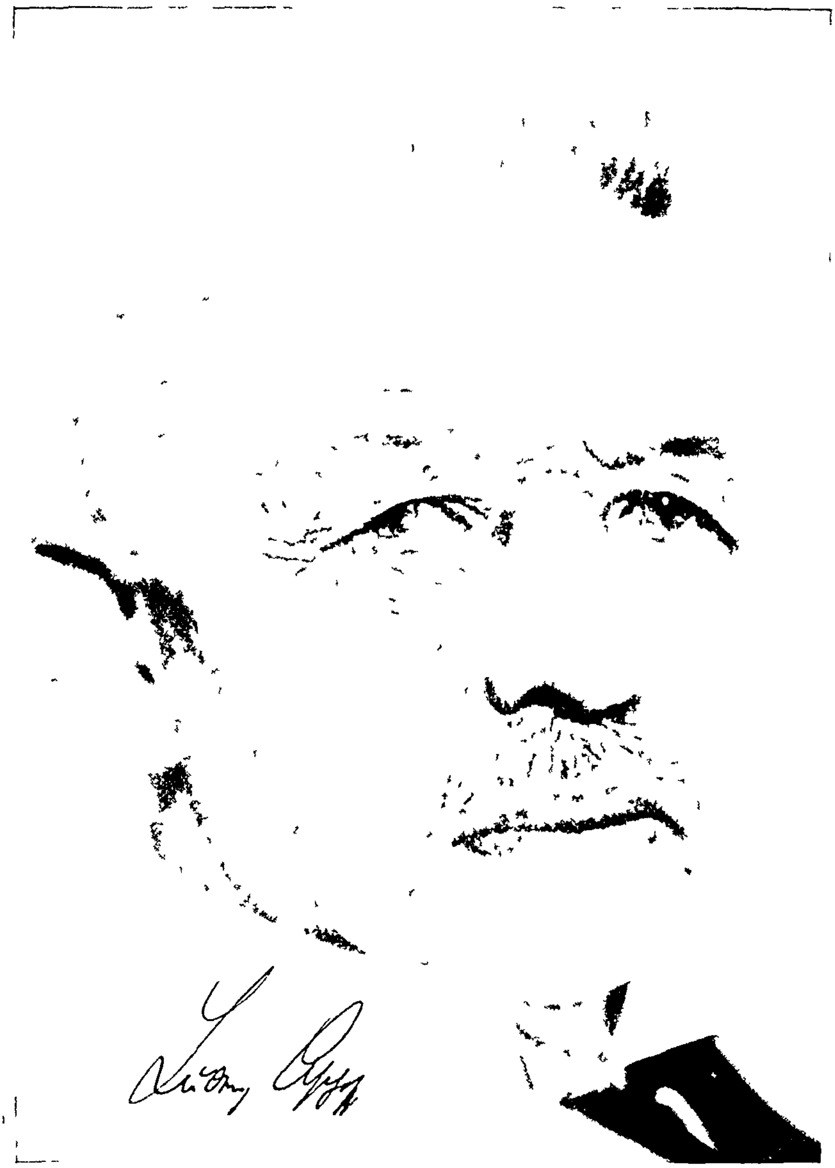
The constancy of form of the curve of an individual is noteworthy. The form appears to change so slightly with the elapse of time that with a little practice one can again recognize the electrocardiogram of the same individual.

We close this paper with a comment relative to the small, irregular, undulations which are visible in most electrocardiograms, varying in amplitude from 0.1 to 0.5 mm. or more, but which, as in Nr. 1 (case of Mr. Ad.), are entirely absent. That these undulations are not produced by vibrations of the floor or other defects in technique is apparent, as they are faintly visible in the normal curve free from vibrations, and are nearly always observed at the end of every series of electrocardiograms. They must, therefore, result from electromotive effects within the human body, and the question is raised, whether their origin is found in the activity of the heart or perhaps, in the activity of other organs. We may expect that a contemplated investigation of this problem may bring forth a definite solution.

1904

LUDWIG ASCHOFF

DESCRIPTION OF RHEUMATIC MYOCARDITIS



LUDWIG ASCHOFF

(Courtesy Journal of Pathology and Bacteriology)

LUDWIG ASCHOFF

(1866- —)

LUDWIG ASCHOFF was born in Berlin on January 10, 1866, the son of a distinguished physician He began the study of medicine at the University of Bonn, supplemented his training at the Universities of Berlin and Strasbourg and returned to Bonn, where he received his degree of Doctor of Medicine in 1889

During his years as a medical student he had decided to become a pathologist and, therefore, shortly after he received his degree he accepted a position as assistant in pathology at the Pathological Institute of the University of Strasbourg, of which Friedrich von Recklinghausen (1833-1910) was director Aschoff served under this great teacher for two and a half years He then accepted a position as assistant to Johannes Orth (1847-1923) in the University of Gottingen, and soon become associate professor After nine years he became ordinary professor of pathology at the University of Marburg In 1906 he was appointed the director of the Pathological and Anatomical Institute in Freiburg im Breisgau, where he now resides

The literature of pathology has been greatly enriched by Aschoff's many original contributions He published as his dissertation an enlightening paper on the effects of the Staphylococcus pyogenes aureus on inflamed tissues In 1900 he published, with Harvey Russell Gaylord (1872-1924), the "Course in Pathologic Histology" In 1902 his important study on Ehrlich's side-chain theory and its application to artificial immunization was published From 1903 until 1909 Aschoff contributed many articles on the physiology and pathology of the heart During this time he described, in collaboration with S Tawara, the conductive system of the mammalian heart He also published two more outstanding contributions which forever will be associated with his name The first of these was his description of the rheumatic nodule, of which we print a translation, the second was his study of the double refractile substances

In later years Aschoff elaborated on the pathology of the conductive system of the heart A most interesting recent contribution on a phase of this subject was his paper[1] entitled "The Significance of the Conducting System in the Determination of Congenital Heart Lesions" This was read in 1937, when he was president of the International Association of Medical Museums

The studies of Aschoff on the conductive system of the heart, on the double refractile substances, and his researches with Rinya Kawamura (1879-) on cholesterol metabolism finally led to an understanding of the pathologic processes in arteriosclerosis This, in turn, led to his description of the reticuloendothelial system

Aschoff's institute soon became the center of pathologic research and his ideas and contributions later in life are elaborated on in the many papers which he published in collaboration with his students Among his students were many Japanese The paper on the reaction of the reticuloendothelial system to dyestuffs, which he published with K Kiyono, his researches in collaboration with Suzuki concerning the accumulation of vital stain substances in the kidney, and many other contributions—with

[1]Aschoff, Ludwig The significance of the conducting system in the determination of congenital heart lesions, J Tech Methods 17 95-96, 1937

Skokichi Nagayo (1866-1910), Kusama, Mitsutano Ogata, Kawamura, and Tawara—are proofs of the great sphere of the influence of Aschoff which dominates Japanese pathology and medicine

In later years Aschoff published his original contributions concerning the origin of the monocytes, the formation of gallstones, and the problems associated with the formation of bilirubin His work on the pathology of the stomach, the pathogenesis of tuberculosis, and his studies concerning goiter are of particular significance

In 1908, and later, in 1930, Aschoff made extensive investigations concerning the bacterial flora found in the normal and the inflamed appendix In 1912 he published, in collaboration with B von Beck, Oscar de la Camp, and Bruno Kronig, his important contribution on thrombosis

The conception of general pathology, its correlation with physiology and clinical medicine, is probably best illustrated in his "Textbook of Pathologic Anatomy,"[2] first published in 1909 Today, after having appeared in several editions, it still is one of the most comprehensive and important surveys in its field

In 1924 Aschoff published some of his contributions on pathology, including some of his American lectures,[3] delivered earlier in 1924 On June 29, 1938, he delivered the Finlayson Lecture[4] before the Royal Faculty of Physicians and Surgeons of Glasgow The subject of his address was the history of the circulation This was exceptionally well told

In 1936, on the occasion of his seventieth birthday, Aschoff was the recipient of a tremendous ovation from his students and friends Today he is justly looked on not only as the greatest living pathologist, whose name will ever be linked with his many original observations, but also as one of the most impressive personalities in the past and present history of medicine

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[2] Aschoff Ludwig ed *Pathologische Anatomie Ein Lehrbuch für Studierende und Ärzte,* bearbeitet von E Albrecht (et al) Jena G Fischer 1909 2 v

[3] Aschoff Ludwig *Lectures on Pathology,* New York 1924 Paul B Hoeber 365 pp

[4] Aschoff L The History of the Circulation, Glasgow M J 130 53-75, 1938

CONCERNING THE QUESTION OF MYOCARDITIS*

By

MR. L. ASCHOFF

Marburg

GENTLEMEN Those who foster the myogenic theory of heart muscle contraction as originated by the Leipzig physiologic and clinical groups, also foster the present day teaching of myocarditis The myogenic theory is supported by such good evidence that it occupies a firm, unassailable position in opposition to the neurogenic theory and pathology and pathologic anatomy is absolutely necessary in the consideration of this subject The myogenic theory states that the automatic movement of the heart muscle lies wholly in the muscular tissue and that the existing nerve centers in the heart muscle, as well as those nerve paths leading to the heart, only serve to mediate reflexes from the heart to the rest of the vascular system and the entire organism, and in a reversed manner Through these means the automatic movement of the heart can be strongly influenced, yes, be entirely stopped, but one could never be assured that the heart would be stimulated through nerve impulses or that its automaticity could be assured through its nerve centers A marked alteration in the movement of the heart, even stand-still of the heart, can occur in two entirely different ways, through abnormal stimulation of the nerve innervation of the heart and through changes in the heart muscle itself It is therefore necessary in every case of severe disturbance in the action of the heart to conduct a thorough investigation, both of the nervous system, that is, the centers in the medulla oblongata, the nerves, and heart ganglia, and of the heart muscle itself, in order to venture a safe opinion regarding the ultimate cause of the heart disturbance, if the heart and its nerve apparatus can be implicated as the origin of this disturbance and not the rest of the vascular system A systematic investigation of the nerve apparatus is, however, confronted by great difficulties, as is the systematic investigation of the heart muscle according to the method inaugurated by Krehl, which, owing to its tediousness, was only carried out in a few definite forms of heart disturbances Thereby, especially through the surprising results confirmed by Krehl and Romberg the weakness of the heart muscle following

*Zur Myocarditisfrage, Verhandl d deutsch path Gesellschaft, 8 46-51, 1904
Translated by F A W

the protracted course of scarlet fever, diphtheria, typhoid fever, *et cetera,* marked changes occur in the form of interstitial inflammation which appears to satisfactorily explain the heart injury rather than the necessity of reverting to disease of the nervous system. Likewise, the important question of why the heart muscle in valvular insufficiency ultimately fails in spite of its hypertrophy, was apparently completely solved by the Leipzig clinicians, through the demonstration of an interstitial myocarditis.

Plainly the myocarditis with valvular insufficiency appeared to be the best evidence for the myogenic theory of automatic contraction of the heart muscle, and in this connection, changes in the heart nerves and ganglia are hardly to be expected and are not disclosed, and the interstitial changes appear sufficiently widespread to explain the weak and irregular contraction of the heart. Is this actually so? I will not fail to mention that this observation was not confirmed by other investigators. And when one studies the work of the Leipzig school more closely, one finds that interstitial myocarditis is only accepted in a part of the cases, and in these, often to a small extent. Further proof seemed desirable. I had Dr. Tawara examine 150 hearts without special selection; of these, sixty were examined in a manner approaching the systematic form of the method of Krehl. Among the latter were three cases of acute nonulcerating endocarditis, and eight cases of chronic endocarditis with marked stenosis and insufficiency of the aortic and mitral orifices. In addition, there was one case of pure acute myocarditis. All these cases clearly belong to the group of rheumatic diseases. In five cases, articular rheumatism had certainly occurred. It is only regarding those cases with myocardial changes associated with valvular insufficiency that I will briefly discuss here. The very time-consuming investigation of the remaining hearts is not yet completed.

Of these, two hearts hardened in formalin were selected, and sections obtained from the apex, the anterior and posterior papillary muscles, the posterior wall of the left ventricle, the septum, the papillary muscles, and the conus of the right ventricle, the right and left auricle, and the sections were stained by the various staining methods (van Gieson, Weigert's stain for elastic fibers, polychromic methylene blue, pyronine, incidentally, also according to the Gram method). In addition, corresponding areas were investigated by frozen sections stained for fat with hematoxylin-Sudan.

We were enabled through this comprehensive and somewhat tedious method to recognize all the changes in the heart substance, the muscle fibers, likewise the fibrous tissue and elastic fibers, the cellular elements of the connective tissue in their form and distribution. The result of this investigation is primarily a confirmation of the views of the Leipzig

school regarding the general occurrence of interstitial change in the heart muscle associated with valvular insufficiency. It, however, broadens the concept of the Leipzig followers, insofar as it permits us to more accurately establish the historic coordination of the products of inflammation, and thereby to find peculiar nodules, which appear to be specific for rheumatic myocarditis. These nodules were plentiful and clearly delineated in only two cases of recurrent endocarditis, but corresponded exactly in their location to the cellular growth in the other cases. They regularly occur in the neighborhood of small or medium-sized vessels, and most frequently were present in the vicinity of the adventitia. Or there existed simultaneously a disease of all the vascular layers, such as is described in arteritis nodosa. The aforementioned nodules are unusually small, mostly submiliary, and originate by the conglomeration of large elements, with one or more abnormally large indented or polymorphic nuclei. The arrangement of the cells frequently occurs in the form of a fan or a rosette. The periphery is formed by the large nuclei, the center by the paler or colorless appearing necrotic mass of confluent cell protoplasm. By cursory examination, the fan formations slightly resemble the necrosis of gout with a peripheral cell mantle, as is so frequently observed in the gouty kidney. The rheumatic nodules are not to be confused with tubercles or foreign body cells with more uniformly formed nuclei, but are of a configuration that more nearly resembles the larger nuclear elements in certain sarcomas or the infiltrations in pseudoleukemia. In all events, the nodules do not exclusively consist of such large nucleated cells, but also small and large lymphocytes, and polymorphonuclear leucocytes force themselves a short distance between the large cells of the periphery, or form a peripheral zone, and from there, irregular projections may extend far into the connective tissue partitions. In these richly cellular projections are found isolated cells with large nuclei, with all the transitions to a simple large leukocytoid element, which are even found in a normal manner in the neighborhood of the smallest vessels and appear very distinct in all inflammations. These leukocytoid elements are the large cells already described by Hayem and Romberg, the genesis of which, however, remains uncertain to them. From these large cells, which are the inflamed swollen adventitial cells of the vessels, the giant cell-like large nucleated element arises, these appear singly or are collected in nodules, and give the rheumatic cellular infiltration its peculiar configuration. It may be further stated that the number of eosinophilic nucleated cells in these nodules is extremely small. While in the one case the structure of the nodule gives the impression of a fresh cellular infiltration, in another case a partial or complete fibrous replacement of the nodule is evident. I have already remarked that this nodule formation shows an identical localization and proves its close relationship to that of the interstitial inflammatory

changes in other cases. Likewise, the nodule-free cellular infiltration in other cases of valvular insufficiency may be histologically recognized by its similar conformity, insofar as the large adventitial cells are concerned, in intimate mixture with large and small lymphocytes and lobulated nuclei, in part the eosinophilic nucleated leukocytes contribute to the perivascular collection.

I investigated this peculiar nodular formation in somewhat more detail, and I found a very similar formation in a case of acute interstitial myocarditis in which no trace of acute or chronic endocarditis existed and except for visible skin hemorrhages, no visceral changes were found. The illness, three weeks after a healed hand injury, led to sudden death. The pattern of this myocarditis is almost identical to that in the case described by Askanazy in a Konigsberger dissertation. Insignificantly small areas of necrosis were formed in the heart muscle fibers, pronounced punctate and cordlike cellular infiltration of the entire heart substance, predominated by eosinophilic nucleated leukocytes, and great numbers of Charcot-Leyden crystals in the rich, cellular, partly necrotic areas. In this case, in addition to the eosinophilic nucleated leucocytes, it is easily understood that the infiltrating adventitial cells also play an important role. Furthermore, isolated plasma cells, large and small lymphocytes, and also occasional fibroblasts, were found. But noteworthy of mention were the sparsely developed large cell nodules similar to those previously described. In this case in which sepsis was presumed to have existed, all investigations for the demonstration of microorganisms were failures, in blood cultures from the vessels *intra vitam* from the heart during postmortem examination and in the stained sections. I believe, therefore, that in this case we are concerned with an infection similar to that occurring in rheumatic endocarditis, the causative agent of which is difficult to determine. Experiences during my stay in Gottingen have shown me that such cases of acute interstitial myocarditis, with a preponderant proportion of eosinophilic nucleated leukocytes and evident Charcot-Leyden crystals, are not so uncommon with and without existent valvular insufficiency. The condition is frequently linked with trauma, overexertion, contusion, and injury to the outer layer of the skin. How the entrance or the localization of the specific virus ensues, is not entirely clear. As we have the large cell nodule formation only in rheumatic endocarditis, and never in the typhoid heart, diphtheria heart, *et cetera*, we believe it permissible to conclude that it is especially characteristic of rheumatic myocarditis. On the other hand, the investigations of Dr. Tawara have necessitated restrictions in the teachings of the Leipzig school. It was, namely, in spite of the comprehensive systematic investigations, that the described interstitial changes in the other cases were so slight, that the symptoms of heart failure observed clinically could not be associated with the changes in the connective tissue struc-

ture In five cases a connective tissue infiltration was virtually absent, in three cases only old, healed scars were found Here, all important sections of the heart were examined, thus, in these cases interstitial myocarditis cannot be the cause of the heart failure, which was very pronounced in just two cases in which clinical data were available (the material came largely from the outside)

According to our experience, certain allowances must be made for those cases with valvular insufficiency, for the diminished efficiency of the heart muscle, at least in a large number of cases, may be sought for elsewhere than in progressive connective proliferation This can only be sought in the muscle substance of the heart, or, if one does not wish to accept the myogenic theory, in the ganglia Therefore, in all the cases, Dr Tawara took sections through the left auricle in such a manner that larger and smaller ganglion cell groups were accessible and included in the investigation

To consider first the ganglion cells in spite of staining with polychromic methylene blue or pyronine, apparent changes were so seldom detected, and particularly in cases of pronounced heart failure, that the source of the muscle insufficiency must originate elsewhere The tigroid substance was chiefly well maintained, likewise the nuclei and the nucleoli, only seldom was early disintegration of the chromatin substance evident, which could be interpreted as occurring after death The occasional marked swelling of the basket cells was striking In every case, fresh inflammatory or older scars were not detected in the connective tissue of the ganglia, as would be anticipated in the course of subacute or chronic inflammation of the heart valves

There remained, then, only the muscular substance Its significance was revealed by the comprehensive, outstanding, assiduous monograph of Albrecht, which appeared during our investigation He considers the universal enlargement of the heart muscle and the ensuing degeneration the same as a parenchymatous inflammation, and reiterates the view that the diminution in the functional activity of the hypertrophied heart muscle can be explained in a different manner from that expressed by the Leipzig school He based his views chiefly on the fact, that with the evident hypertrophy, no increase in the effective contraction substance occurred, it was not in the fibrils, but only in the sarcoplasm, that the nuclei assumed the form of "Leistenkerne" already described by Romberg, that these progressive changes result in a regressive form of granular degeneration of the protoplasm, a shrinkage or swelling of the nuclei, until finally, instead of single muscle cells, only nuclei-containing pigmented collections of protoplasm alone remain The connective tissue infiltration is somewhat secondary

Unfortunately, the investigations of Dr Tawara were in no manner able to support these findings Without regard to Albrecht's basic error

that conjoined muscle cells actually exist while all these striations only represent differences in form and condition of special contraction phenomena, it is easy to conclude that in the hypertrophied fiber segments in addition to the increase of sarcoplasm, there also occurs thickening of the fibrils, that the peculiar elevated nuclear formations of Romberg are not pathologic, but already occur early in youth, or at least exist in the physiologic *Anlage,* but only appear more distinctly in the hypertrophied muscle bundles. All of Albrecht's depicted nuclear formations are also found in hearts in all decades which are not hypertrophied, except for the fact that the nuclei are smaller and the formation is less distinctly seen. The observation that the nuclei of the heart of a youth or an adult under normal circumstances occur in oval or rodlike formations, with smooth surfaces, must finally be relegated. They only exist in this condition in the newborn and young child. Even in an older child, more so in an adult, the nucleus assumes a flat form on both sides, which like a flat ring is bent over the surface with points and extensive projections. The greatest diameter of the nucleus is frequently at right angles to the longitudinal direction of the fibers. The size of the nucleus and its chromatin content are very variable. Definite degenerative nuclear changes do not exist, not considering the disintegration of the nuclear substance. One can only distinguish between simple, hypertrophic or atrophic nuclei, but, nevertheless, all transitions are present and the ranges are difficult to determine.

On the other side, concerning the views brought to the foreground by Albrecht regarding the changes of the nuclear degeneration of the muscle fibers, which occur independently of embolic processes or inflammatory necrosis, it may be said that we have not observed these changes. Vacuolar nuclear degeneration and fatty infiltration also play a large role in hypertrophied hearts, and surely impair heart efficiency, but do not lead to an appreciable degree of loss in muscular substance. But they can also exist in hypertrophied hearts that lag in their work, fail, or possess such slight reserve that it does not suffice for the explanation of the heart failure. I surely do not wish to consider the purely agonal occurrence of fragmentation if I do not consider pigmentation, the beginning of which may already be found in the first decade of life.

When, therefore, we previously could find no histologic pattern for the failure of the muscle mass, it was necessary to consider that not the muscle mass as such, but only a certain part, was injured, namely, the so-called atrioventricular bundle, which gives automaticity according to the myogenic theory. On that account, Dr. Tawara studied serial sections from the region of the membranous portion, together with parts of the auricular and ventricular septa in a series of eight cases, of which three belonged to the above-mentioned category. He was enabled to prove that the atrioventricular bundle in all cases corresponded to the

descriptions of Kent, His, and Retzer Contrary to Retzer's findings, it was apparent that in the newborn as well as in the adult, a marked difference existed between the strength of the fibers and the form of the nuclei in the fibers of the atrioventricular bundle and the ventricular muscle At its entrance in the auricular septum, the atrioventricular bundle forms a dense texture in the shape of a node The result of the investigation was that in no case, as in those three cases of rheumatic endocarditis that are of special interest to us in this consideration, were appreciable changes discovered

Therefore, on the grounds of Dr Tawara's investigation we must conclude (1) that the enlargement of the heart muscle in valvular insufficiency produces a true hypertrophy, and (2) that inflammatory changes do not have the described significance and do not explain the decrease in efficiency of the hypertrophied muscle, but that the heart muscle weakens and we are unable to observe by means of our present-day laboratory aids, degenerative changes of a greater extent in the muscular substance And a circumscribed lesion of the atrioventricular bundles does not enter into the consideration of the cases thus far studied

1907

ARTHUR KEITH AND MARTIN FLACK

DEMONSTRATION OF THE SINOAURICULAR NODE (NODE OF KEITH AND FLACK)

Arthur Keith

SIR ARTHUR KEITH

(Courtesy Journal of Anatomy)

SIR ARTHUR KEITH

(1866- —)

SIR ARTHUR KEITH, senior author of the classic we are reproducing, was born in Old Machar, Aberdeen, Scotland, on February 5, 1866. He is the son of John and Jessie Macpherson Keith.

Arthur Keith studied at the University of Aberdeen, where he received the degrees of Bachelor of Medicine and Master of Surgery, with first class honors, in 1888. In 1894 he received the degree of Doctor of Medicine, with highest honors, from the same university. In 1894 he also qualified for membership and received his fellowship in the Royal College of Surgeons. He also studied at University College, London, and the University of Leipzig.

In 1899, Keith married Cecilia, daughter of Tom Gray, the artist. She died in 1934.

Early in his career as an anatomist, Keith developed a profound interest in anthropology. In 1896 appeared his first work, entitled "An Introduction to the Study of the Anthropoid Apes." Other works by Keith about anthropology are "Ancient Types of Man" (1911), "The Antiquity of Man" (1915, second edition, 1925), "Nationality and Race from an Anthropologist's Point of View" (1919), "Concerning Man's Origin" (1927), and "New Discoveries Relating to the Antiquity of Man" (1931).

Keith also contributed two books on Darwinism: "Religion of a Darwinist" (1925) and "Darwinism and Its Critics" (1935). The work for which he is probably most widely known is his "Human Embryology and Morphology." This was first published in 1901, and the fifth edition of this work appeared in 1933. He also is author of "The Human Body" (1912) and "Engines of the Human Body" (second edition, 1925).

To the field of orthopedics, Keith contributed an important historical work entitled "Menders of the Maimed" (1919). In this volume he traced the development of the anatomic and physiologic principles underlying the treatment of injuries to muscles, nerves, bones, and joints. At the time of the publication of this work, Keith was conservator of the museum and Hunterian professor of the Royal College of Surgeons of England. He was appointed to this position in 1908 and continued in it until his retirement in 1933. Before this time, and at the time he and Flack were working on the node, Keith was lecturer in anatomy, in London Hospital Medical College.

In 1921 Keith was created a baronet. He had served as secretary of the Anatomical Society of Great Britain from 1899 to 1902, and as president from 1913 to 1917. When, in 1916, the "Journal of Anatomy" became the property and official organ of the Anatomical Society, he was appointed acting editor. He held this position for seventeen years and the unqualified success of this authoritative journal is the result, to a great extent, of Sir Arthur's indefatigable labor and skillful guidance.

Sir Arthur has held many enviable positions. In 1913, he was elected a member of the Société d'Anthropologie de Paris. He served as Fullerian professor of physiology at the Royal Institution from 1917 to 1923. He was secretary of the Royal

Institution from 1917 to 1923, and in 1926 and 1927 was treasurer From 1930 to 1933 he was rector of the University of Aberdeen He has received the honorary degree of Doctor of Laws from the Universities of Aberdeen, Birmingham, and Leeds In 1920 he received the degree of Doctor of Science from the University of Durham In 1923 he received the same degree from the University of Manchester and in 1930 the University of Oxford gave him another He is also a fellow of the Royal Society of London

At present, Sir Arthur is master of the Buckston Browne Research Farm in Downe, Farmsborough, Kent, England It was not far from that farm, near Flack's native village of Borden, in a small cottage in an orchard, that Keith and Flack, during their college vacation, carried out their valuable study on the node [1]

MARTIN WILLIAM FLACK

(1882 1931)

MARTIN WILLIAM FLACK was born in the village of Borden in Kent, England, in 1882 He received his preliminary education at the Great Yarmouth and Maidstone Grammar Schools Later he matriculated at Keble College, Oxford, where he studied general science as well as medicine Among his teachers were Arthur Thomson (1858-1935), Francis Gotch (1853-1913), and John Scott Haldane (1860-1936) In 1905, Flack received the degree of Bachelor of Arts from Oxford University

Flack completed his medical studies at the London Hospital, where he had been awarded the Price Scholarship There he came under the direction of Sir Leonard Erskine Hill (1866-), who then was a lecturer in physiology, and Sir Arthur Keith, who was a lecturer in anatomy, in the London Hospital Medical College Arthur Keith[2] at that time was studying the nervous and muscular structures of the heart and invited young Flack to work with him

In 1906, Tawara[3] announced his discovery of a system of conducting musculature between the auricles and ventricles of the mammalian heart Keith and Flack verified the truth of Tawara's discovery and set out to trace the evolution of the auriculo-ventricular connecting system They also desired to ascertain if, in the region wherein the beat of the heart was believed to begin—namely, at the termination of the superior vena cava in the right auricle—there existed tissue of the nature of the node which Tawara discovered at the beginning of the auriculoventricular bundle They found in all mammalian hearts, at the expected site, a collection of peculiar muscular tissue This tissue, they named the sinoauricular node, inferring from its position, and from its resemblance to the tissue at the beginning of the ventricular bundle, that this node was concerned in the inception of the heart beat This important classic, published in 1907 in the "Journal of Anatomy," we are happy to reproduce for our readers At a later date, according to Keith, Lewis demonstrated the exact site at which the normal heart beat originates and proved that the site coincides with the position of the sinoauricular node

Flack at the time of his clinical studies at London Hospital acted as assistant demonstrator of physiology under Sir Leonard Hill He assisted Hill in research problems which were mainly concerned with circulation and respiration This experience was of great benefit to him later, when he studied the medical problems of aviation

[1]Personal communication from Sir Arthur Keith
[2]See preceding biographic sketch
[3]Tawara, S *Das Reizleitungssystem des Saugetierherzens,* Jena 1906, Gustav Fischer

MARTIN FLACK

Photograph by Russell and Sons

In 1908, Flack received the degrees of Bachelor of Medicine, Bachelor of Surgery, and Master of Arts from Oxford University That same year he was married to his cousin, Cecile Cooper Four children, two sons and two daughters, were born to them

In 1909, Flack received a Radcliffe Travelling Fellowship This gave him the opportunity of studying on the Continent, study for which he had long had a desire At Bern, Switzerland, he worked under Hugo Kronecker (1839-1914) and Asher, studying the nerve supply to the thyroid gland He also spent some time at Liège, Belgium, where he studied the heart under the great Léon Frédéricq (1851-1935), who had been chosen by Théodor Schwann (1810-1882) to become professor of physiology in the University of Liège in 1881 at the age of thirty, and who proved the existence of fibrinogen in the blood plasma

Flack returned to London and continued as demonstrator in physiology in Hill's department at the London Hospital He collaborated with Hill in many investigations and later, in 1909, became co-author with Hill of the work "A Textbook of Physiology"

In 1913, Hill was asked by the Medical Research Council to establish a department of applied physiology He chose Flack to assist him in this undertaking The World War began the next year and in the early part of the struggle, Flack lent his services to the army With Mervyn Gordon he combated the epidemics of cerebrospinal fever then current As a result of this service he was breveted lieutenant colonel in the Royal Army Medical Corps, and on the formation of the Royal Air Force Medical Service he was made wing commander and director of medical research

The World War demonstrated the need for the investigation of the oxygen starvation of airplane pilots at high altitudes, and Flack was chosen for this work He not only made an inquiry into the need of oxygen by flyers at high altitudes, but also developed a series of comprehensive tests to determine the physical fitness of applicants for the air service The results of these important studies were made the subject of his Milroy Lectures, which he delivered before the Royal College of Physicians in 1921 In 1923 Flack was promoted to the rank of group-captain

Group-Captain Flack represented the British Medical Services at the San Antonio meeting of the Association of Military Surgeons in 1924 He also attended the meeting of this organization in Washington, D C, in 1930

Flack suffered for many years from a rheumatic affection of the heart but in spite of this physical handicap achieved great ends His death on August 16, 1931, was the result of a septicemia

THE FORM AND NATURE OF THE MUSCULAR CONNECTIONS BETWEEN THE PRIMARY DIVISIONS OF THE VERTEBRATE HEART*

By

ARTHUR KEITH, M.D. (Aberd.), F.R C.S. (Eng.)

Lecturer in Anatomy, London Hospital Medical College

And

MARTIN FLACK, B.A. (Oxon.)

Assistant Demonstrator of Physiology, London Hospital Medical College

Introductory—In a paper contributed to *The Lancet* some months ago (5), the writers confirmed and extended the discovery of Tawara (7), viz that there is within the mammalian heart a system of peculiar musculature (the a-v system) which, beginning as a small root (the *Knoten*) in or near the base of the interauricular septum on the right side, eventually spreads out in an arborescent form beneath the endocardium of both ventricles, its final twigs becoming everywhere continuous with the ordinary musculature of the ventricles It seemed to us essential to examine other regions of the heart for such peculiar musculature Moreover Wenckebach (8) has demonstrated by exact clinical methods that a delay may occur in the conduction of the cardiac impulse from sinus to auricle This fact stimulated us to investigate fully the nature of the muscular connection between the sinus and the auricle, which has already been partly described by Wenckebach in the human heart

In this paper therefore the writers propose to deal with the results of an extended inquiry, made with three objects in view —

I To ascertain the extent, nature, and position of the muscular connection or connections between the primary divisions of the heart in all classes of the vertebrate kingdom

II To seek in the sinus, auricle, and bulbus cordis for a differentiation in form and structure of a system of muscle fibres corresponding to that now known to exist in the ventricle in short, to ascertain whether the musculature in which the heart-impulse is held to arise, and by which it is conducted, differs in form and structure from that which is mainly contractile in nature

III To trace the evolution of the a-v muscular system, as found in the human heart, from the simpler and more definite form seen in the heart of fishes

*J Anat & Physiol 41 172-189, 1907

Material—It is important that those who may consult this paper should know the exact material used by us in this inquiry, and its method of preparation In the appended list of material we do not include the numerous hearts which have been dissected by knife and forceps, but only those which have been examined by a series of microscopic sections

List of Material

Fishes—Eel, dog-fish, salmon

Amphibia—Frogs (3)

Reptilia—Lizard (species unknown), tortoise, turtle

Birds—Sparrows (2), goldfinch

Mammals (other than human)—Mole, porpoise, dolphin, kangaroo, wallaby, whale (*B musculus*), mouse, shrew-mouse, rat (2), kitten (2), ram, pig, cart-horse, pony, foetal gibbon

Human—Embryos (2), normal hearts, malformed hearts, and fifteen hearts having definite pathological lesions

Method of Preparation—For macroscopic specimens for dissection a modification of Kaiserling's method was used The great advantage of this method is that the natural colour of the muscle fibres returns after fixation, thereby rendering easier the dissection of the different systems of muscle fibres The procedure is as follows —

(1) The heart must be well washed in running water for 12 hours prior to fixation, and the cavities stuffed with tow or cotton-wool

(2) The specimen is then fixed in the following solution

Formalin, 200 c c
Water, 1000 c c
Potassium nitrate, 15 grams
Potassium acetate, 30 grams

In this solution it remains at least 24 hours, and longer if it be large, hard, or tough

(3) Specimen placed in 80 per cent spirit until its colour returns

(4) Kept in equal parts of glycerine and water

In the preparation of microscopic specimens stages (3) and (4) are omitted After (2) the specimen, or the desired parts of it, is well washed in running water It is then transferred to spirit (24 hours), next alcohol in stages from 70-100 per cent for 24-48 hours, then in xylol until clear, and finally embedded in paraffin We have found that the process of embedding is much facilitated by exhausting the incubator By this means clear, firm blocks with no trace of air-bubbles are obtained The blocks were cut in the main at 10μ, except when it was desired to study the minutest structure of the specimen, when they were cut from 4μ to 7μ in thickness, according as the nature of the tissue permitted The

sections were stained by Ehrlich's acid haematoxylin and Van Gieson's stain, dehydrated, and mounted in Canada balsam It is important to overstain with haematoxylin, otherwise the nuclei will not be well seen, owing to the decolorising action of the second stain

Literature—With regard to literature, we have been unable to find any previous paper approaching the nature of our research The writings of Gaskell (2), MacWilliam (6), and Engelmann (1) have proved of great service We accept the teaching (1) that the heart's impulse is conducted by the cardiac muscle tissue, (2) that normally the impulse arises in the musculature of the sinus, setting the heart's rhythm, and then passes to the auricle and ventricle, finally reaching the bulbus cordis

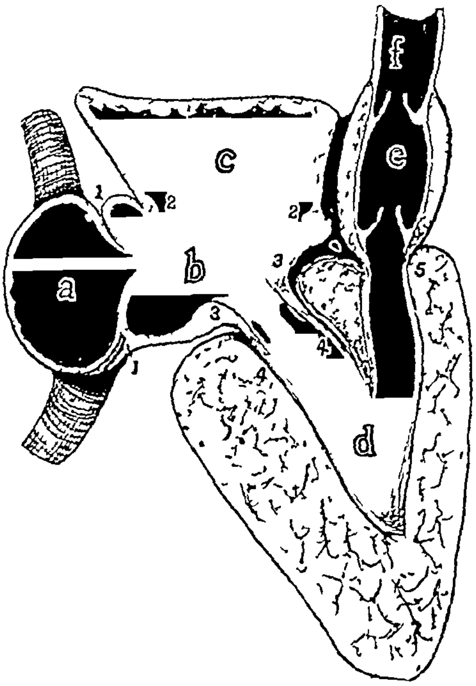

FIG 1—Diagram of a generalised type of vertebrate heart—combining features found in the eel, dog fish, and frog—showing the primary cardiac chambers and their lines of union

a, sinus venosus and veins *b*, auricular canal *c*, auricle, *d*, ventricle, *e*, bulbus cordis *f*, aorta, 1-1, sino canalar junction and venous valves, 2-2, canalo auricular junction, 3-3, annular part of auricle, containing special muscle fibres, 4-4, invaginated part of auricle, 5, bulbo ventricular junction By the longitudinal fibres lining the ventricle there is a connection between the annular fibres of the auricle and the bulbus musculature

The Primary Divisions of the Vertebrate Heart—Before proceeding to describe the muscular connections between the primary divisions of the heart, it is necessary for us to define exactly what we regard as such They are well seen in the generalised diagram (Fig 1) There are five primary divisions of the heart —

(1) The sinus venosus (*a*)
(2) The auricular canal (*b*)
(3) The auricle (*c*)
(4) The ventricle (*d*)
(5) The bulbus cordis (*e*)

There are four junctional lines —

(1) *The Sino-canalar*, marked by the venous valves, the free margin of the valves forming the boundary-line between the cavity of the sinus and cavity of the auricular canal (*1-1* in Fig 1)

(2) *The Canalo-auricular*, marked by a thickening of the musculature round the ostium of the auricle, situated on the dorso-lateral wall of the auricular canal (*2-2* in Fig 1)

(3) *The Canalo-ventricular*, marked in the mammalian heart by the auriculo-ventricular valves, the free margins of which separate the cavity of the auricular canal from that of the ventricle (*4-4* in Fig 1)

(4) *The Bulbo-ventricular*, situated at the junction of the ventricle and bulbus (*5* in Fig 1)

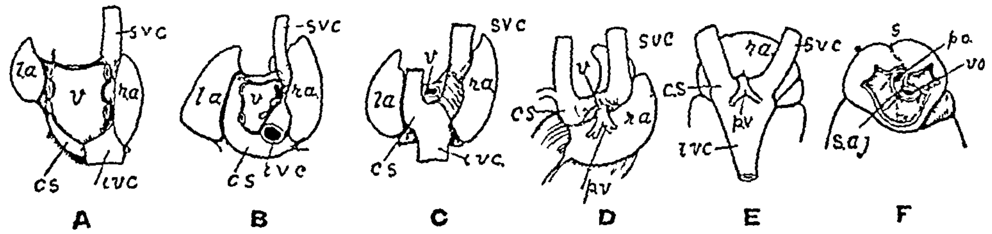

FIG 2 —Series of diagrams to illustrate the parts in the human heart corresponding to the sinus of the primitive heart

A, dorsal view of auricular part of the human heart B corresponding view of wallaby s heart, C corresponding view of the heart of a child in which the lungs were fused and the vestibule of the left auricle consequently unexpanded D, corresponding view of the heart of a malformed fœtus in which the inferior vena cava was absent E, corresponding view of the heart of the frog (Gaupp) F corresponding view of the heart of the frog, the interior of the sinus venosus being exposed to show that the pulmonary veins open within the sino auricular junction In this figure *p o*, orifice of pulmonary veins *v o*, orifice of sinus venosus *s*, attachment of inter auricular septum, *s a j*, sino auricular junction *v*, vestibule *r a*, right auricle *l a*, left auricle, *s v c*, superior vena cava, *c s*, coronary sinus, *i v c*, inferior vena cava

The Primary Divisions and Junctional Lines in the Mammalian Heart — Our knowledge of the heart has been derived in great part from experiments made on the simpler hearts of the eel, frog, and turtle, in order to transfer accurately that knowledge to the mammalian, and more especially to the human heart, it is necessary to identify in them the primary divisions which are seen so clearly in the simpler hearts We propose, therefore, in the first place, to identify the five primary divisions above mentioned in the human heart

The Sinus Venosus of the Human Heart —In Fig 2 is shown a series of illustrations of views of the sinus venosus in various hearts, the view represents the venous or auricular end of the heart, looked at from the dorsal side The sinus (see *E*) is formed by the union of three great vessels—the right duct of Cuvier (*rt sup v c*), the left duct of Cuvier (*lft sup v c*), and the inferior vena cava (the hepatic vein of fishes) In the human and in the mammalian heart, the musculature of the auricular canal has grown over and submerged the greater part of the sinus (*o*, Fig 3), two parts only are left exposed on the surface of the heart—(1) the musculature of

the superior vena cava, (2) the musculature of the coronary sinus (the representative of the left superior vena cava) (see Fig 2 *A, B C*) But if a section be made across the line at which the sinus becomes submerged (the stria terminalis of His), a second or deep stratum of musculature is seen (beneath *o*, Fig 3), this probably belongs to the sinus venosus, since it extends beneath the endocardium of the auricle, from the position of one venous valve to that of the other Besides these three definite remnants of the sinus musculature, there is also the musculature—or part of it—in the Thebesian and Eustachian valves, these being remnants of the right venous valve There is often also to be found a thin muscular layer along the lower border of the fossa ovalis, it occupies the position of the left

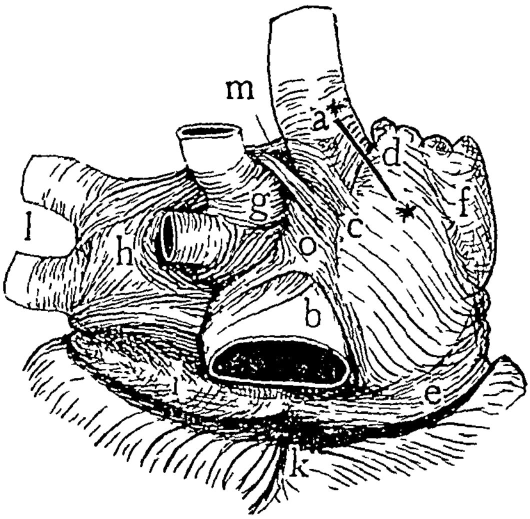

FIG 3 —The auricular part of the human heart from behind, showing the musculature of the termination of the great veins

a superior vena cava surrounded by musculature derived from the sinus, *b*, inferior vena cava *c*, to the right of the sulcus terminalis, above *c*, sinus fibres cross the sulcus to join auricle proper, *d*, at sino auricular junction, where peculiar musculature is found most abundantly, *e*, annular fibres of auricle, *f*, appendix, *g*, fibres passing from interauricular septum to vestibule of left auricle between the two left pulmonary veins, *h*, vestibule *i*, coronary sinus, showing continuity of fibres with right and left auricles, *k*, base of ventricles at interventricular sulcus, *l*, left pulmonary veins, *m*, constant band passing from sinus musculature to vestibule of left auricle, *o*, muscle of auricular canal submerging sinus, ** represent line of section of fig 6, A

venous valve, and is probably derived from it Thus, the chief remnants of the sinus venosus have to be sought for in the right auricle of the human heart Its musculature is represented by —

(1) The termination of the superior vena cava
(2) The coronary sinus
(3) The submerged stratum
(4) The remnants of the venous valves

It may be, however, that there are also remnants of the sinus in the left auricle of the human heart In Fig 2, *F*, it is seen that in the heart of the frog the musculature of the sinus at the sino-auricular junction (*s a j*) includes within it the orifice of the pulmonary veins This is also seen in the heart of the malformed foetus, Fig 2, *D* It is possible, therefore,

that, as the part of the auricular canal (*v*) which is to become the vestibule of the left auricle expands, a part of this sinus musculature is also involved in the process, and may persist in the left auricle of the human heart around the orifices of the pulmonary veins

In a part or in all of this sinus musculature the heart rhythm is believed to be initiated

The Auricular Canal of the Human Heart—In the simplest form of heart the auricular canal, which joins the sinus venosus to the ventricle, is differentiated into three parts (see Fig 1)—(i) a basal part (opposite the auricle), (ii) an annular part (*3-3*), (iii) an invaginated or intraventricular part (*1-1*) The invaginated part forms an isolated layer beneath the auriculo-ventricular valves, its musculature becoming continuous with that of the ventricles near the apices of the valves (Fig 1) Only a small part of this musculature remains in the human heart, it forms the a-v

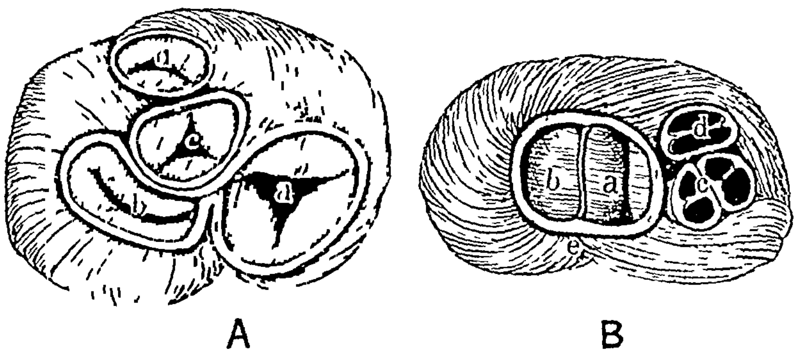

FIG 4 —To illustrate the infolding and modification of the auricular ring in the mammalian heart

A, base of the ventricles of human heart B, base of the ventricles of heart of turtle *a*, right auriculo ventricular orifice *b*, left auriculo ventricular orifice, *c*, orifice of aorta *d*, orifice of pulmonary artery *e*, posterior part of the auricular ring, which becomes infolded in the mammalian heart The stippled part near *e* represents the only part which remains undifferentiated in the mammalian heart

bundle This we shall treat of more fully later on The annular part of the canal—the "auricular ring," as we shall term it in this article—has in the human heart become submerged in the auriculo-ventricular groove just above the base of the auriculo-ventricular valves (*c*, Fig 3) Only one essential change has taken place This can best be clearly explained by the help of a figure

In the reptilian as in the amphibian heart (Fig 4, *B*) the annular part forms a simple ring, the interauricular septum lies within it, separating the right from the left a-v orifice But in the mammalian heart the simple annular form has been lost, owing to the extension of the bases of the ventricles backwards under the basal wall of the auricular canal, the annular part has been folded as shown in Fig 4, *A*, so that the mesial folded part has now come to rest upon the upper or auricular margin of the interventricular septum From this superventricular fold of the annular ring begins the a-v bundle (stippled in Fig 4, *A*)

The basal part of the auricular canal is best defined by explaining its origin The auricle or auricles are outgrowths from the dorsal wall of the auricular canal (see Fig 1), the ventral wall remains unspecialised as the basal part The basal part, it will be seen, is continuous with the sinus venosus, with the ostium of the auricle, and with the auricular ring From a physiological point of view the basal part of the auricular canal is most important, since both Gaskell and MacWilliam found that it was a path of conduction from the sinus to the ventricle, so that a sino-ventricular rhythm could occur It is therefore interesting to see whether the possibility of this rhythm remains in the mammalian heart In the human, as in the mammalian heart, the basal wall has become profoundly modified by two great cardiac transformations which have occurred with the evolution of the pulmonary system These changes are (1) the formation of the interauricular septum, (2) the formation of a vestibule to the left auricle (Figs 2 and 3) The basal wall has supplied a large part, if not the whole, of these two structures As the fibres of the lowest part of the interauricular septum come into intimate relation with the annular ring, it will be seen that it is therefore possible for a sino-ventricular rhythm to occur in the human heart Indeed, a layer of longitudinal muscle fibres passes directly from the superior vena cava into the auricular septum, and thus reaches the musculature from which the a-v bundle commences The musculature of the three parts of the auricular canal is represented in the human heart thus —

(1) The basal part by the interauricular septum and by the vestibule of the left auricle

(2) The annular part by the circular fibres surrounding the ostia above the bases of the auriculo-ventricular valves The annular fibres also descend for some distance on the septal cusp of the tricuspid valve

(3) The invaginated part of the a-v bundle

The Auricles of the Human Heart—In the fish's heart, the common auricle forms a well-demarcated outgrowth on the dorsal wall of the auricular canal The ostium, by which it opens on the canal, is surrounded and indicated by a thick circular ring of musculature (Fig 1, *2-2*) In the mammalian heart, the development of the interauricular septum and of the vestibule of the left auricle from the basal wall has led to a division of the auricle and to a wide separation of its two parts (see Fig 3) However, in the mammalian, and especially in the human heart, a prominent ridge of musculature, commencing in the right auricle immediately in front of the termination of the superior vena cava and seen on the roof of the left auricle, still unites the two auricles, and represents the original continuity of the two chambers (see Fig 7, *A*)

Thus in the right auricle of the human heart there is musculature derived from three sources—(1) from the auricle proper, (2) from the auricu-

lar canal, (3) from the sinus venosus In the left auricle the musculature arises from (1) auricle proper and (2) auricular canal All these parts are in the freest muscular continuity

The Ventricle of the Human Heart —It is unnecessary in this place to discuss the correspondence of the common ventricle of the lower forms with the divided ventricular chamber of the higher forms They are developed as outgrowths from the ventricular segment of the primitive cardiac tube, the part which remains undisturbed between the outgrowths forms the interventricular septum The upper margin of the septum represents the least disturbed part of the lumen of the primitive tube, on it lies the a.-v bundle

The Bulbus Cordis of the Human Heart —This fifth division of the heart is well marked in the primitive forms (Fig 1, *e*) It is generally supposed to be absent in the mammalian heart, but recently Greil (3) and one of the authors (4) has shown that this is not so, the infundibulum of the right ventricle represents practically the whole of this cavity The musculature of the bulbus has become replaced entirely or for the greater part by that of the ventricle

The Musculature of the Sinus Venosus and of the Sino-auricular Junction —Having thus sketched out, perhaps too briefly, the primary divisions of the heart, we now propose to describe the musculature of the sinus venosus and its connections with the other parts of the heart, more particularly in relationship to two points in physiology (1) The rhythm of the heart begins in the sinus does its musculature or any part of its musculature show any peculiar differentiation in connection with this function? (2) What are the muscular connections of the sinus, are they restricted so that a sino-canalar or sino-auricular "block" may occur, as is supposed by Wenckebach, or are they so wide and diffuse that such a block is inconceivable from an anatomical point of view? These two matters we shall discuss in relationship to the human heart, using our comparative material only in so far as it throws light on the questions we discuss

Taking the latter question first, we may say at once that the musculature of the sinus is freely continuous with that of the auricular canal and of the auricle An impulse arising in the sinus musculature around the termination of the superior vena cava (Fig 3, *a*) may spread directly (1) into the musculature of the interauricular septum, and thus to the network in which the a.-v bundle begins (Fig 5, *b*), (2) to the vestibule of the left auricle (*m*, Fig 3), and to the auricle proper along the interauricular bridge (Fig 5, *c*), (3) to the auricular canal of the right auricle, (4) to the right auricle proper (Fig 5, *d*) If the impulse commences in the coronary sinus, then it may spread directly (1) to the vestibule of the left auricle, (2) to

the annular fibres of the left auricle, (3) to the annular fibres of the right auricle (Fig 3, *i*) Indeed the higher one ascends in the vertebrate scale, the less becomes the amount of the sinus musculature, but the greater the closeness of its connection with the canalar and with the auricular musculature It therefore appears to us that the sino-auricular "block" cannot be due to an anatomical lesion of a narrow bridge of fibres, but must arise from the depression, probably of vagal origin, of the muscular tissue in this region

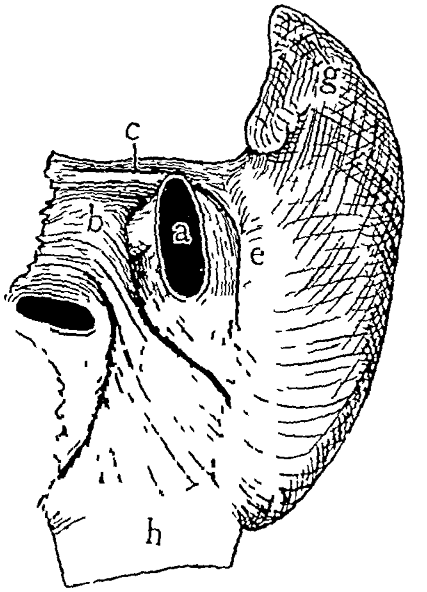

FIG 5 —Right auricle of human heart viewed from above, to show connections of musculature at the termination of the superior vena cava

a, superior vena cava cut across, *b*, vestibule of left auricle, fibres are seen to enter the interauricular septum from the superior vena cava, *c*, sinus musculature of superior vena cava passing to left auricle (*m* in fig 3), *d*, sinus musculature of superior vena cava crossing sulcus terminalis to right auricle, *e*, sino auricular junction, *f*, musculature of interauricular septum submerging sinus *g*, appendix, *h*, inferior vena cava, *i*, septal fibres passing on to vestibule of left auricle below orifice of pulmonary vein

In Fig 6 we give two sections of the sino-auricular junction [1] *A* is that of the human heart made across the sulcus terminalis in the position shown in Fig 3, *B* that of the heart of the turtle The venous valve (see Fig 6, *B*) at the sino-auricular junction is seen to be really a fold of the cardiac tube, the musculature of one side of the valves is derived from the sinus—that of the other is continuous with the auricular musculature At the free margin of the valve the sino-auricular muscle is continuous (Fig 6, *B*, *3*) A certain amount of fibrous tissue belonging to the epicardium is enclosed within the folds of the valve, in this an artery is frequently present By the musculature of the valves an impulse may be freely distributed in the musculature of the auricular canal and of the auricle proper, for at the upper and lower angles at which the valves unite their musculature

[1] We use the term 'sino-auricular' in preference to "sino-canalar" because, although a true sino-canalar junction exists on the dorsal side in the most primitive hearts (see Fig 1), yet in all but these the part of the canal between the sinus and the auricle disappears, and the dorsal junction becomes really a sino-auricular junction Moreover, as the term auricle is usually applied in the mammalian heart to the parts representing both auricular, canal and auricle proper the term 'sino-auricular is the more appropriate

spreads out and joins freely with that of the auricular division of the heart (Fig 6, *B, 4*) In the mammalian heart a distinct remnant of the sino-auricular junction, so well shown in more primitive hearts, can be recognised (Fig 6, *A*) In the human heart, as in most mammalian hearts, an artery or arterial circle lies in the junction (Fig 6, *A, 2*), the artery is surrounded by fibrous tissue in which are numerous peculiar muscle fibres, some nerve cells and nerve fibres The nerve cells and fibres we find from dissection to connect with the vagal and sympathetic nerve trunks which form so rich a plexus and exert so powerful an effect at this junction The

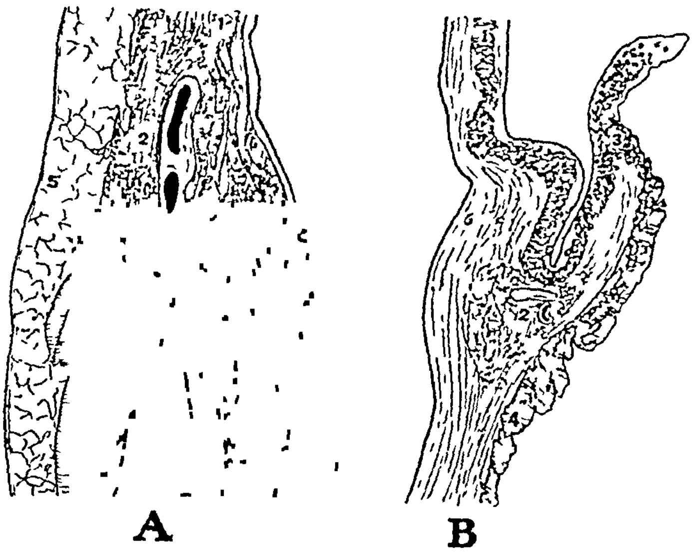

FIG 6 —A, sino auricular junction in human heart (position indicated in fig 3), B, sino auricular junction in turtle's heart The figures represent corresponding parts in the two hearts

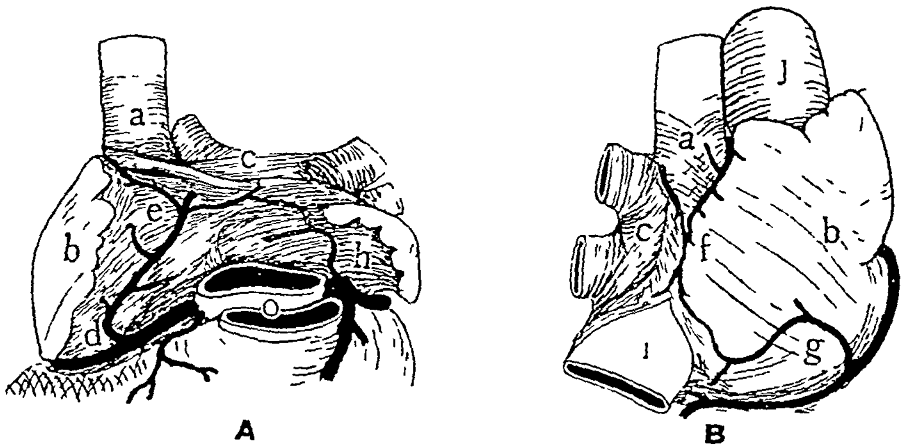

FIG 7 —Showing blood supply of the musculature of the sino auricular junction

A, aorta and pulmonary arteries removed, exposing auricles from the front, B, right auricle from the side *a*, superior vena cava *b*, appendix, *c*, vestibule of left auricle *d*, *artery arising from right coronary* and passing to sino auricular junction at *e* the artery divides one branch passing in the junction in front and the other in septum behind superior vena cava *f* union of two branches above mentioned in sulcus terminalis *g*, anastomosing branch from right coronary artery *h*, anastomosing branch from left coronary artery, *i*, inferior vena cava, *j*, aorta, *o*, aorta and pulmonary artery

musculature of the superior vena cava becomes continuous with that of the auricle and of the auricular canal both on the outer and inner side of the artery

Our search for a well-differentiated system of fibres within the sinus, which might serve as a basis for the inception of the cardiac rhythm, has led us to attach importance to this peculiar musculature surrounding the artery at the sino-auricular junction (Fig 6, *A*, 2) In the human heart the fibres are striated fusiform, with well-marked elongated nuclei, plexiform in arrangement, and embedded in densely packed connective tissue—in fact, of closely similar structure to the *Knoten* The amount of this musculature varies, depending upon how much of the sinus has remained

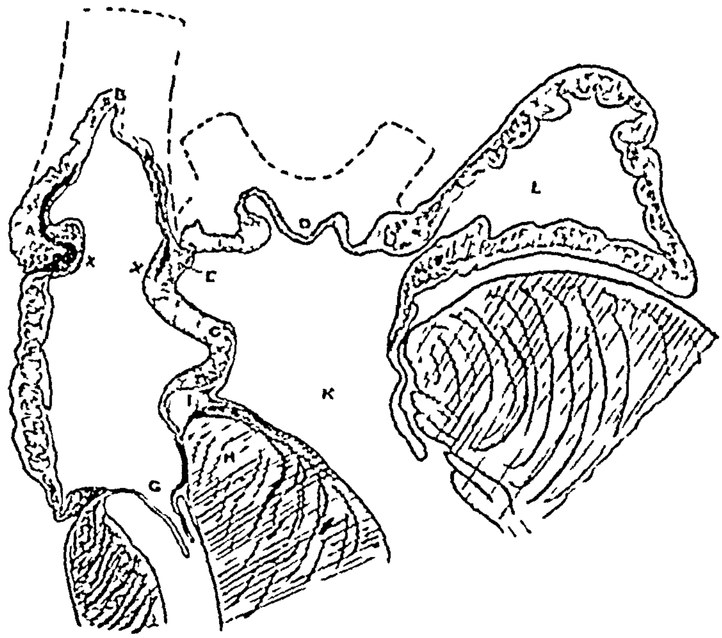

Fig 8 —Coronal section of the mole's heart, showing position of musculature at sino auricular junction

xx, junction of superior vena cava and right auricle, A peculiar musculature described in paper at sino auricular junction, B, section of wall of superior vena cava the position of the superior vena cava is outlined C, interauricular septum D, vestibule of left auricle the pulmonary veins are outlined E, similar musculature to A lying at junction of superior vena cava and vestibule, F, wall of right auricle G, right auriculo ventricular orifice H, interventricular septum, I, a v bundle similar in structure to A, K, left auriculo ventricular orifice, L, left auricle **, canalo auricular junction

of the primitive type, but in the neighbourhood of the taenia terminalis there is always some of this primitive tissue found Macroscopically, the fibres resemble those of the a-v bundle in being paler than the surrounding musculature, *i e* in being of the white variety They can be dissected out on the superior vena cava in the region corresponding to the right venous valve (*a*, Fig 3), and at the coronary sinus in the interval between it and the inferior vena cava and left auricle (*b*, *i*, Fig 3) Another remarkable point in connection with these fibres is the special arterial supply with which they are provided (Fig 7, *A* and *B*) These arterial branches, as noticed by Wenckebach, embrace the sino-auricular junction It will be seen that they come from both right and left coronary arteries

and form what may be termed the "sino-auricular arterial circle." We might mention also that, in some of the pathological hearts cut by us, sections of this region appeared to show a definite increase in the amount of fibrous tissue present—a fact of considerable importance, since we have found that the fibrous tissue of the *Knoten* and a.-v. bundle is sometimes increased in pathological hearts.

The nature of this remnant is perhaps best exemplified in the heart of the mole (Fig. 8). Here it is seen that at the sino-auricular junction (*A, E*) there is a mass of remarkable tissue. It appears to the eye as a very intimate network of palely stained undifferentiated fibres with a large number of well-stained nuclei. It is totally different from the surrounding musculature, and contains but little fibrous tissue. Although the mass by its connections is undoubtedly muscular, the nerves in the neighbourhood of the superior vena cava appear to come into very intimate connection with it, so much so that we feel justified in stating that a highly differentiated neuromuscular junction occurs at this point. In this heart also the bundle (*I*, Fig. 8) is of absolutely identical structure.

In a section of the heart of the wallaby in this region there is seen under the low power (2″) a mass of fibrous tissue apparently separating superior vena cava from auricle. On closer inspection, however, it is seen that very delicate, palely stained, primitive muscular tissue is enclosed within the fibrous mass.

A section of this junction in the porpoise's heart is interesting. The musculature of the superior vena cava has largely remained primitive in type. The wall of the superior cava consists of alternate layers of fibrous tissue and primitive palely staining fibres. Just at the junction, however, of the superior vena cava and auricle, a network of these fibres and fibrous tissue is formed. In it there is an artery, and two nerve trunks lie close by.

In the dolphin's heart, on the other hand, there is no difference between the greater part of the musculature of the superior vena cava and that of the auricle. But in the region of the taenia terminalis there occurs some loosely-woven fibrous tissue, in the meshes of which are contained an artery and wavy, delicate muscle fibres with well-marked nuclei.

The ram's heart also shows a similar characteristic set of fibres in this region. The tissue in this case shows a marked amount of fibrous tissue loosely interwoven with palely-staining fibres closely resembling those of the sinus of the frog. The remnant also occurs in the hearts of the kitten, rat, and mouse.

From the above it will be seen that the presence of these primitive fibres is remarkably constant. Physiologic experiments have clearly demonstrated that normally the heart's rhythm begins in the neighbourhood of the great veins, and that here nervous influence has a most potent effect (MacWilliam, Engelmann, Hering, and others). The fact, therefore, that there is a constant differentiation of certain fibres in this region, which, moreover,

are in close connection with the nerves affecting the heart's rhythm, leads us to attach great importance to these fibres, and we feel justified in expressing the opinion that it is in them that the dominating rhythm of the heart normally begins

The Canalo-auricular Junction—At the ostium of the auricle in the lower hearts the musculature is directly continuous with that of the auricular canal (Fig 1, *2-2*) In them there is a difference in the type of fibre constituting the two parts, those of the auricle being coarser, more striated, and more deeply stained In the mammalian heart the junction between the parts representing the auricular canal and the auricle proper is also marked by a thickening of the musculature The fusion of the different systems of muscle fibres, however, is for the most part so intimate that it is difficult to distinguish between them We could find no trace of any especially differentiated fibres at this junction

The Canalo-ventricular Junction—This is the junction of the auricular canal with the ventricle It has been described by MacWilliam in the heart of the eel In this heart the auricular ring is connected to the ventricular system by the fibres of the invaginated part of the auricular canal (see *4-4*, Fig 1) This part of the canal shows a differentiation even in the eel Its fibres differ from those of the rest of the canal in being larger, less striated, staining more palely, and possessing a very large distinct nucleus

In the frog there is a similar connection all round the auriculo-ventricular orifice below the base of the a-v valves, but particularly below the auricular septum The fibres of the connection are not differentiated from those of the rest of the canal they are shut off by fibrous tissue from the ventricular system in the upper part of their course, but later on they fuse with the fibres of the innermost part of the ventricular wall The canalo-ventricular junction in the reptilian heart is similar to that of the fish and of the frog

In the mammalian heart the auricular ring and the invaginated fibres become profoundly modified Taking the human heart as a type, we find that the ring can still be traced round the right auriculo-ventricular orifice above the bases of the valves The fibres are no longer isolated, but can be identified by their structure No trace of them can be found in the canal of the left auricle It will perhaps be well to recall the arrangement of the muscular connection between the auricular canal and the ventricle in the human heart The system begins in the *Knoten*, a small mass of interwoven fibres in the central fibrous body of the heart, having slender connections with (1) the musculature of the interauricular septum, (2) the circular fibres of the right auricular canal From this arises the main bundle which passes along the upper border of the interventricular septum below the pars membranacea septi Here it divides into a right and a left division, which pursue a subendocardial course in the right and left ventricles, respectively, and finally fuse with the ventricular muscle The fibres composing the

main bundle, and more especially its arborisations, vary very much in type in the hearts of different species. In some hearts there is a marked difference from the ordinary ventricular musculature. Such is the case in the hearts of the sheep, ox, calf, cart-horse, pony. In these the main bundle consists of long, delicately striated fibres, with large nuclei. The end arborisations consist of fibres belonging to the Purkinje system. In other hearts the fibres of the bundle and its terminal branches are not so well differentiated from the ventricular fibres. This is the case in the whale, kangaroo, wallaby, dolphin, man, rat, kitten, mouse, shrew-mouse, and pig. In these hearts, however, and especially in the first four mentioned, there is still a differentiation of fibre rendering the bundle quite distinct from the ordinary ventricular musculature. The fibres of the bundle are larger, more delicate, less striated, and stain less deeply than those of the ventricle proper. In certain other hearts, namely, those of birds, the authors have been unable to find any differentiation of fibres in the bundle, the guides to it being its position and its definite demarcation by fibrous tissue. In the bird's hearts examined by us, the a.-v. bundle arises from the auricular ring and dives at once into the interventricular septum.

The a.-v. bundle is the sole muscular connection between the auricular canal and the ventricle; there is no direct connection between auricle proper and ventricle in the mammalian heart. It must be admitted, however, that in one case, namely, in the heart of a rat, the auricular and ventricular fibres appear to come into close apposition in the right lateral auriculo-ventricular region, and undoubtedly represent one of the connections described by Stanley Kent. In the heart of the sparrow also there is a similar close apposition of fibres in this region. This close apposition, however, cannot be looked upon as a connection; the a.-v. bundle is to be regarded as the sole connection between the auricular canal and the ventricle.

The Bulbo-ventricular Junction.—This junction is well marked in the primitive hearts (see *5*, Fig. 1). In all a circular groove containing epicardium separates the ventricular from the bulbar musculature, but not completely; the inner or subendocardial layer of ventricular musculature becomes continuous with the bulbar musculature. In the frog's heart this is also the form of connection, but the union is three or four times denser on the dorsal than on the ventral side of the b.-v. junction.

In the mammalian heart the bulbus has become fused with the right ventricle, forming the infundibulum of that cavity. Greil's research on the heart led him to the conclusion that, although the cavity of the bulbus remains, its musculature has been overgrown and replaced by that of the ventricle. The moderator band which passes from the septal wall of the right ventricle to the base of the anterior group of musculi papillares marks the separation of the bulbar part from the rest of the right ventricle. On this band of muscle the right division of the a.-v. bundle descends; that

is, if our identification be correct, the right septal division descends in the position of the bulbo-ventricular junction There can be no doubt, at least, that there is *no* bulbo-ventricular separation of fibres *in* the mammalian heart

The Morphology of the A-V Bundle—This is the third point which we had in mind during this research As the result of our examination of the hearts in different branches of the vertebrate kingdom, we have come to the following conclusions in reference to the morphology of this bundle —(1) The *Knoten* represents the only part of the annular ring of the auricular canal of the primitive heart which has remained undifferentiated in type The rest of the ring has become differentiated and is imbedded in the other auricular musculature as explained above (2) The main bundle and its two divisions represent the remnant of the invaginated portion of the auricular canal

The chief evidence in favour of (1) may be summarised thus —The different position occupied by the *Knoten* in relation to the central fibrous body in the hearts of different animals, *e g* of the sheep, horse, and man, points to the fact that in each a different portion of the auricular ring has remained undifferentiated as the *Knoten* The musculature of the *Knoten* resembles in structure the other portion of the primitive canal which has remained undifferentiated, namely, the remnant at the sino-auricular junction, evidenced especially by the hearts of the mole, rat, and ram Lastly, in the heart of a human embryo (32 mm long) the auricular ring is clearly seen, and the part which is to persist as the *Knoten* is in close continuity with the ventricular musculature The ring in this embryo is at the upper part of the interventricular septum, and its fibres are of exactly the same type as persist in the *Knoten* throughout life

The evidence that the main bundle is the remnant of the invaginated portion of the auricular ring reveals itself as we proceed from the lower to the higher forms In the eel this part of the auricular canal forms the a-v connection, which is all round the auriculo-ventricular orifice In the amphibian and reptilian heart the connection is still around the whole orifice, but it is thickest at the base of the interauricular septum In the bird's heart the connection is comparatively large, and is situated solely at the base of the interauricular septum In the mammalian heart the connection is small, and occupies the upper border of the interventricular septum It is beyond the purpose of this article to discuss the physiological reason for this restriction of the muscular connection between the auricle and the ventricle to a narrow bundle which measures on the average only 1 5 × 0 8 mm in diameter, but its persistence in the position which it occupies becomes intelligible when it is called to mind that the upper border of the interventricular system represents the least-disturbed part of the lumen of the embryonic cardiac tube

Summary

I (a) The muscular connection in the lower hearts between sinus and auricular canal, and in the higher between the parts of the heart representing them, is intimate In the latter, fibres pass directly from this junction to the vicinity of the a.-v bundle

(b) The canalo-auricular junction is marked by a thickening of the heart wall at this point The muscular connection is diffuse In the lower forms there is a difference between the fibres of the two parts, but in higher forms the fusion is so intimate that no difference in the type of fibre can be distinguished

(c) The canalo-ventricular junction decreases in extent from the lower to the higher forms, in the latter it is represented solely by the a.-v bundle

(d) The bulbo-ventricular junction is well marked in the lower hearts In higher forms the ventricular musculature has replaced that of the bulbus

II (a) There is a remarkable remnant of primitive fibres persisting at the sino-auricular junction in all the mammalian hearts examined These fibres are in close connection with the vagus and sympathetic nerves, and have a special arterial supply, in them the dominating rhythm of the heart is believed to normally arise

(b) No special differentiation of fibres was found at the canalo-auricular and bulbo-ventricular junctions

III (a) The *Knoten* is a part of the primitive auricular ring which has remained undifferentiated

(b) The main bundle and its branches represent the invaginated portion of the primitive auricular canal

We wish to express our thanks to Mr Humphrey Neame, and particularly to Mr William Chesterman, for their help in the preparation of the microscopic sections

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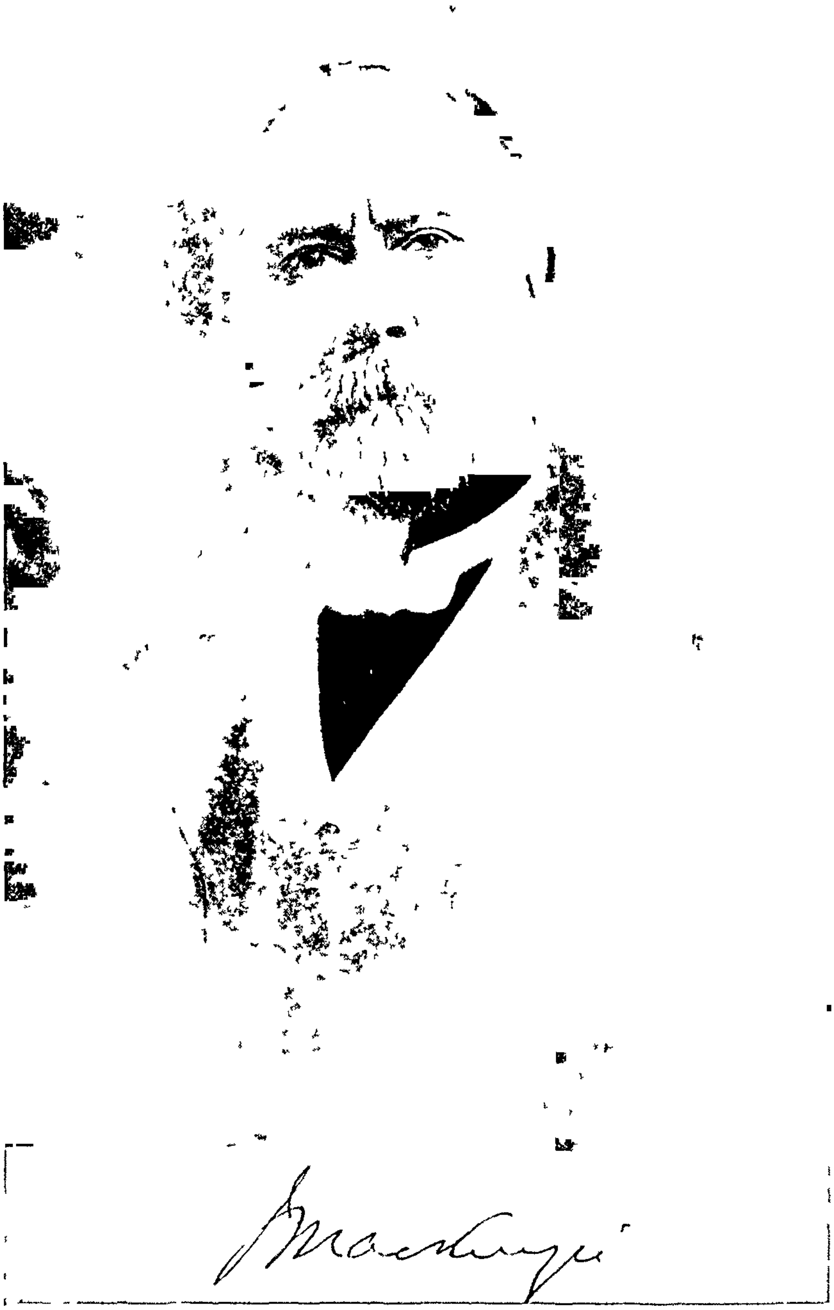
Note—Since the above was written, Dr J Mackenzie has kindly drawn our attention to a recently published paper of Hering's, "Uber die Automatie des Saugethierherzens," in *Pfluger's Archiv*, Bd 116, p 143 It is interesting to note that Hering brings about complete stoppage of the supraventricular parts of the heart by a cut made at the sino auricular junction in precisely the same position as our section (Fig 6, *A*)

1908

SIR JAMES MACKENZIE

DESCRIPTION OF AURICULAR FIBRILLATION



SIR JAMES MACKENZIE
Photograph by Emery Walker

(Courtesy Charles C Thomas)

SIR JAMES MACKENZIE

(1853-1925)

"The Beloved Physician"

—R McNair Wilson

JAMES MACKENZIE was born at the farm of Pickstonhill, Scone parish, Scotland, on April 12, 1853 His father, Robert Mackenzie, and his mother, Jean Campbell Menzies, had moved there from Perthshire Near his birthplace stood the ruins of the abbey wherein the kings of Scotland had been crowned since antiquity

Young Mackenzie was a pupil first at the village school and later at the grammar school at Perth, the latter institution being so old that it was well known in 1153 Mackenzie left school, at his own volition, at the age of fifteen, to become an apprentice in a chemist's shop He served his apprenticeship here for five years, and it was the inspiration derived by contacts with country physicians who came to the chemist's shop to have their prescriptions compounded that caused Mackenzie to decide to study medicine After he had served his apprenticeship his employer offered him a partnership in the shop However, he decided to accept a post in Glasgow as a chemist He remained for a year and his work at Glasgow confirmed his determination to become a physician

Mackenzie was twenty-one years old when he went to Edinburgh to begin the study of medicine in the university of that city He had some difficulty, as he himself said, in passing the examinations of his early years, because his power of memory, on which the examinations depended, was not so good as his power to reason During this period of his life as well as during his earlier academic training at Perth, Mackenzie exhibited a sense of inferiority He always felt himself the "dunce" of the school and even later, when he was awarded three medals for superior clinical work at the University of Edinburgh, he still felt that he was a student of the most inferior grade It is of interest to note how well this sense of inferiority paralleled that of the great American historian, Henry Adams, who twenty years earlier, at Harvard College, felt the same frustration and sense of failure Adams had a sense of frustration and spiritual weariness all his life, but as he is acknowledged the authority in certain aspects of American history, so Sir James Mackenzie has often been acknowledged as one of the greatest physicians of the twentieth century

Mackenzie was graduated from medical school in 1878 The year was a conspicuous one in medical history, for it ushered in the opening stages of the battle which Pasteur and Lister were waging in behalf of the germ theory of disease Mackenzie's application for resident at the Edinburgh Royal Infirmary was accepted and he remained there a year In 1879 Mackenzie was induced by Dr John Brown, his former professor in anatomy, to become an assistant in the firm of Dr William Briggs and Dr John Brown at the Lancashire town of Burnley in England At the end of a year's work he was offered a third share in the practice This he accepted, and the situation was so pleasant for Mackenzie that he remained in Burnley for thirty years

Early in his practice at Burnley, Mackenzie was summoned to help a young woman in childbirth She was his own patient The delivery promised to be easy and uneventful But during the stages of labor a tragedy occurred his patient suddenly

died of heart failure. James Mackenzie, as he turned to break the news of her death to her husband, tasted the bitterest anguish which any doctor can ever experience. He felt responsible for the death of his patient, for there probably had been, long before the time of delivery, some cardiac sign by means of which, had he recognized it, he might have been able to take certain precautions. There and then Mackenzie resolved to specialize in the study of the heart.

He began by studying the mechanism and symptoms which generally are presumed to indicate cardiac conditions in pregnant women. On consulting the literature he found nothing that proved helpful. To find answers to the riddles of the heart, Mackenzie made innumerable studies. In investigating the circulatory conditions before, during, and after pregnancy not only among women who had cardiac disease but also among healthy women, he found changes in the size and position of the heart, murmurs of various types, variations in rate and rhythm, and other departures from what is usually considered the normal. To distinguish types he resolved to "write" the pulses of a large number of women and then to study the tracings with the closest possible attention. At that time there was available the Dudgeon sphygmograph or pulse writer. With the use of this instrument Mackenzie soon confirmed his contention that several different varieties of irregularity occurred in the tracings. But what did these different waves in the tracings signify? Which were truly dangerous ones, and which were innocuous? There did not seem to be an answer.

Another fact confused him. He found out that the pulsations of the vessels of the neck were just as varied as were the waves of the pulse. Mackenzie felt that if tracings of the pulsating vessels of the neck could be obtained, some factors might be correlated that would aid in the solution of the problems which beset him. He therefore modified the Dudgeon apparatus and made the first crude tracings of the pulsations of the vessels of the neck.

Mackenzie's work on the tracings was interrupted by a most pleasant occasion. His marriage to Miss Frances Bellamy Jackson took place on September 13, 1887. The honeymoon was spent in Italy. The marriage itself was a happy one, Lady Mackenzie later declaring that "No woman ever had such a generous love and companionship given her."

As has been suggested, Mackenzie's difficulty in employing the mechanical registration of pulsations was to interpret the various waves he was able to inscribe. At first he attempted to determine the answers to these enigmas by questioning the leading physiologists of his time, but they could not answer his queries. Some time later Mackenzie devised a new, portable recording instrument. Using the radial pulse as a standard and a pill-box for the drum he was able to construct an apparatus which he carried around in his pocket. About this time the ink polygraph[1] and the electrocardiograph made their appearance. However Mackenzie for several years preferred to use his miniature instrument because he believed it was simpler and better adapted for general use. Later, when he found out that the signs he had discovered with his various instruments were also detectable by the finger of the physician, he used the instruments less frequently than before. Mackenzie resented the attitude of the medical profession in too completely accepting the ink polygraph and later the electrocardiograph. He believed that physicians were more interested in the exactness of mechanical devices than in the ends for which these mechanical devices were constructed.

Earlier, Mackenzie had discovered a method of determining how two of the heart's four chambers were acting at any given moment. He had also made the discovery

[1]Later Mackenzie with the help of a Mr. Shaw, a watchmaker of Padiham, perfected the ink polygraph which is today known the world over as Mackenzie's Ink Polygraph.

that pregnant women tended to have irregularities of the pulse Suddenly the idea occurred to him to determine what the various chambers of the heart were doing when that organ was beating irregularly When his next patient presented himself with a marked irregularity of the heart he was able to prove that in some cases of irregular action, the ventricle contracted sooner than normal The auricle, in maintaining its normal rate and rhythm, actually beat after the ventricle Later, Mackenzie demonstrated that this form of irregularity, the extrasystole, was in itself of no serious consequence to the patient Although identification of the extrasystole was in effect a negative discovery, it was most important It was proof that many patients presenting themselves with this type of cardiac irregularity need not be alarmed and that they could lead normal lives

In about 1890 Mackenzie's attention was directed to another group of peculiar irregularities A woman whom he had first seen in 1880, when she was suffering from an attack of rheumatic fever, again presented herself for treatment He discovered that her heart had been damaged, and that the damage was the result of the attack of rheumatic fever in 1880 and subsequent attacks in 1883 and 1884 A presystolic murmur was audible Narrowing of the mitral valve continued Mackenzie maintained careful observation of this patient, noticing that in 1898, with alarming rapidity, his patient grew worse An amazing thing occurred at that time The presystolic murmur vanished! After considerable thought, Mackenzie came to the conclusion that the auricles of the heart had ceased to beat, and that it had been the beat of the auricles forcing the blood through the narrowed passage in the heart that had caused the presystolic murmur A year later the patient died, and on examining the heart Mackenzie verified his belief He found the auricles to be enormously distended and greatly weakened He gave to this type of irregularity the provisional name "paralysis of the auricle" It is now known as "auricular fibrillation," because of Mackenzie's research and the subsequent researches of a favorite pupil, Sir Thomas Lewis

Mackenzie noted these conditions among a number of patients and his recognition of the disappearance of auricular contractions instituted a landmark in the history of medicine It was the central feature of much subsequent work on the elucidation and grouping of the various anomalies of the rhythm of the heart It further prepared the way for Mackenzie to establish, together with Cushny, the conditions necessary for efficient digitalis therapy The results of Mackenzie's early observations appeared in his "Study of the Pulse" (1902)

The unique experiences of Mackenzie in cardiology have been admirably set forth in his monumental work, "Diseases of the Heart," of which the first edition was published in 1908 His careful study of semeiology appeared in 1909 Another important work, "The Future of Medicine," appeared in 1919 This volume is mainly autobiographical and expresses the wisdom which made him famous Besides revising his small and large books on the heart, Mackenzie in 1923 contributed a superb monograph on the disease of which he was destined to die, "Angina Pectoris" Another important work, "The Basis of Vital Activity," was completed shortly before his death in 1925 It was not published, however, until 1926

After practicing medicine at Burnley for nearly thirty years, Mackenzie went to London in 1907, at the age of fifty-four In London, although he earned only about $600 during his first year there, he soon became an outstanding Harley Street consultant He continued his studies and somehow found time to write several books which we have mentioned Within a short space he was elected a member of the Royal College of Physicians He was further honored by a fellowship in the Royal Society, and in 1915 he was knighted In 1918 he retired from active practice

The next year he established at St Andrews in Scotland the Institute for Clinical Research[2] Mackenzie defined the object of the Institute's work as "the prevention of the diseases that are common among the people" The reports of the Institute have been regarded as most valuable contributions in the systematic study of symptoms and disease

Shortly after coming to St Andrews, he was stricken by angina pectoris Although he knew of his condition, he continued to work unceasingly at the Institute He revised his great work on "Diseases of the Heart," lectured in London, Edinburgh, Dundee, and Aberdeen He wrote several articles for medical journals and continued working on his several books

His final months were severely painful, and he suffered numerous cardiac attacks He died on January 26, 1925, foretelling the period of his death with uncanny certainty Some weeks before he died, he directed Dr John Parkinson to examine his heart after his death After Mackenzie's death his brother, Sir William Mackenzie (now Lord Amulree), corroborated this request The examination was performed by Dr John Parkinson, assisted by Dr J W Linnell and Dr David Waterston The latter wrote an account of the observations, which we reprint

[2]Now known as the James Mackenzie Institute of Clinical Research

CHAPTER XXX

AURICULAR FIBRILLATION[*]

The importance of recognizing auricular fibrillation.—The most important of the continuous abnormal rhythms is that which is due to fibrillation of the auricles. The recognition of this condition and the symptoms associated with its presence, is the most important discovery yet made in the domain of the functional pathology of the heart, and few physicians are aware of its significance. The symptoms directly due to auricular fibrillation, and the symptoms of heart failure induced by this condition, are so clear and definite, that we have little difficulty in recognizing this condition as a distinct clinical entity. Its recognition is not of mere academical importance, but is of the greatest practical value, for when we recognize the various symptoms, they afford us grounds for a sure diagnosis, a safe prognosis, and for a rational line of therapy in a large proportion of cases of serious heart failure. The great frequency of its occurrence renders it imperative that all practitioners should become familiar with its symptomatology, for 60 or 70 per cent of all cases of serious heart failure met with in practice owe the failure directly to this condition, or have the failure aggravated by its presence. Some of the symptoms have been overlooked in the past, while the significance of others has not been appreciated. Moreover, the response of hearts affected with auricular fibrillation to remedies differs so much from the response of all other forms of heart action, normal and abnormal, that the recognition of its characteristics materially alters the views universally held as to the action of drugs upon the heart.

Type of case showing auricular fibrillation.—Before setting out in detail the features characteristic of auricular fibrillation, it might be convenient to appreciate the kind of case which shows this condition. The most common evidence is an irregular action of the heart of a very disorderly kind. It is that form of irregularity so frequently met with in the elderly, and in patients with hearts damaged by previous rheumatic infection. In the latter class, the association of irregular hearts with mitral stenosis has long been recognized, and, on account of this association, the irregular pulse is sometimes described as the "mitral pulse," with which all clinicians are familiar.

While senile and rheumatic hearts are those most frequently affected by this condition, there are numerous cases in which auricular fibrilla-

*Sir James Mackenzie first described this condition in 1908, in the first edition of his book, *Diseases of the Heart*. We have chosen to reprint from the third edition of this work published in 1914, pp. 211-236.—F. A. W. and T. E. K.

tion occurs, where there is no history of rheumatism, and at an age when senile changes are not present in a marked degree. Auricular fibrillation is present in the great majority of cases with dropsy, or dyspnoea due to heart failure, where there is an irregular pulse. Thus, in Withering's report of cases to whom he administered digitalis successfully, published in 1784, where the symptoms of heart failure were associated with an irregular pulse, and in whom digitalis acted in a special manner, there can be but little doubt that these were cases of auricular fibrillation. Other cases are found recorded as *delirium cordis*. In those predisposed to auricular fibrillation, violent exertion may induce the condition, and many of the recorded instances of *heart overstrain* afford excellent examples of auricular fibrillation, and of the heart failure which accompanies it.

Personal experiences in the recognition of auricular fibrillation.—My attention was first directed to this condition as a separate and definite entity about 1890. I had been endeavoring to discriminate between the different forms of irregular heart action, and it occurred to me to employ the jugular pulse as an aid. By this means I was able to separate the great majority of irregularities into definite groups, according to the mechanism of their production, as revealed by simultaneous records of the jugular and radial pulses. There was one group which showed a distinct difference from all others, by the presence of the ventricular form of the venous pulse. I was at a loss to understand the nature of the heart's action in these cases, and as I found them very frequently among people with a history of rheumatism, I determined to watch individual cases with rheumatic hearts, to see when this irregularity arose, and when the auricular venous pulse changed to the ventricular. The individual recorded as Case 48 came under my care in 1880, suffering from an attack of rheumatic fever. I examined her at intervals until her death in 1898. Up to 1897, her heart was regular, except for occasional ventricular extra-systoles. Her jugular and liver pulses were always of the auricular form (Fig. 119 and 120). There was a well-marked presystolic murmur. She became very ill in 1897, with a rapid and irregular heart. When the heart slowed down after a partial recovery, I found that the jugular and liver pulses were of the ventricular form (Fig. 121), that the presystolic murmur had disappeared, and that the heart was irregular, in other words, all evidences of auricular activity had disappeared. From this date onwards, I was able to confirm these observations, and add to them other cases which showed waves due to the auricle, in jugular and apex tracings before the heart became irregular, and their disappearance when the heart became irregular. Thus, I established that all the positive evidences of auricular activity, capable of being revealed by clinical methods, showed the cessation of auricular action with the onset of this irregularity. For many years, I speculated as to the causes of

auricular fibrillation As the auricle was found distended and thin-walled at the post-mortem examination I came to the conclusion that the disappearance of the signs of auricular systole was due to the auricle having become distended atrophied and paralysed This view I put forward in a book on the pulse which I published in 1902 Shortly after this was published I had a series of cases some of which I had watched for years and at the post-mortem examinations the auricles were not thinned but were hypertrophied With this fact before me I saw that

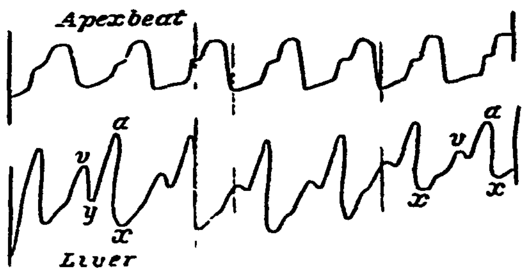


FIG. 119 The liver pulse shows a well-marked wave (*a*) due to the auricle (Case 48 1892)

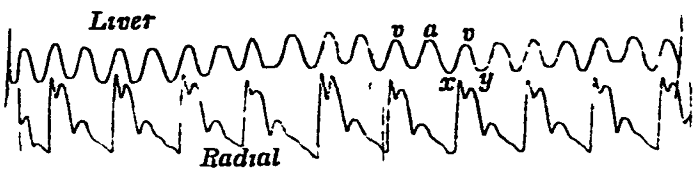


FIG 120 There is still a well-marked wave in the auricle (Case 48 1897)

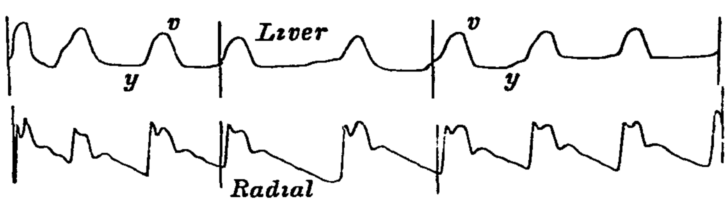


FIG 121 Showing the irregular rhythm, characteristic of auricular fibrillation When compared with Fig 119 and 120, it will be seen that there is no auricular wave in the liver pulse, and the heart's action is irregular (Case 48 1898)

my previous explanation could not be correct, for the fact that the auricles were hypertrophied, indicated that they must have contracted during the years that I had watched them, and when there had been an absence of all signs of auricular activity As it was clear that the auricles could not have contracted during the normal period—that is to say immediately before ventricular systole—the only alternative I could see was that they contracted during ventricular systole As, in the meantime, I had studied several hundreds of cases and had seen this condition start under a variety of circumstances, particularly in individuals with frequent extrasystoles, I put forward the view that ventricles and

auricles contracted together, and assumed that the stimulus for contraction arose in some place that affected auricles and ventricles simultaneously As at this time I could not conceive of any other possibility to explain the facts, I suggested that the stimulus for contraction arose in the auriculo-ventricular node, and I called the condition "nodal rhythm," under which name the clinical aspects of auricular fibrillation are described in the two editions of this book, the first being published in 1908

With the advent of the electrocardiograph, we obtained a more accurate method of recognizing the contraction of the chambers of the heart When electrocardiograms were taken of the cases that I had called nodal rhythm, my clinical observations were verified, inasmuch as no evidence of the normal auricular systole was found In cases where the heart periodically became disorderly in its rhythm, and where I was able to demonstrate that the auricular form of venous pulse was present with the regular heart action, and the ventricular form during the period of irregular action, the electrocardiograms also showed evidence of auricular contraction during the normal period of the heart's action, and a disappearance of the normal auricular activity during the period of irregular action, fully confirming the observations I had made on nodal rhythm

The attention of other observers had also been arrested by some of the clinical features of this condition Thus Hering, in 1903, separated from among other irregularities the irregularity peculiar to auricular fibrillation, and called it the *pulsus irregularis perpetuus* He was mainly concerned with the physiological aspect of the subject, and did not recognize the full clinical picture, with the disappearance of all signs of auricular activity Many other observers had noted the "positive" venous pulse, and in attributing it merely to tricuspid incompetence they had failed to appreciate its real meaning, and so missed the significance of its appearance

Although the disappearance of the auricular contraction was the feature that puzzled me in these cases, I realized that my explanation of it, as being due to synchronous contraction of auricles and ventricles, was far from being established, and I endeavored to interest others in the subject, who might investigate the matter by experimental methods, and find out, if possible, what the auricle was doing Cushny was the first to suggest that auricular fibrillation might be a factor of clinical importance, and in 1906 he and Edmunds drew attention to the resemblance of the radial tracings in a case of paroxysmal irregularity in the human subject to the tracings from a dog, in which they produced experimental fibrillation of the auricles On reading this communication, I was struck with the idea, and on a visit Professor Cushny paid to me in Burnley in 1906, he discussed with me the probability of auricular fibrillation being the cause of the irregular

heart action in certain cases of "nodal rhythm," and he agreed that certain small waves, which I had recognized in the jugular pulse of one case (Fig 122), were due to the fibrillation of the auricle

I published, in 1907, tracings with this explanation, but I failed to appreciate the real significance of what auricular fibrillation was, I thought it only a passing event, and I practically gave up the idea that it was at the bottom of these cases that went on for years Lewis had been pursuing an inquiry clinically and by experiment into the nature of cardiac irregularities, and had produced experimental fibrillation in the dog In 1909 he took graphic records of the venous and arterial pulses With the onset of

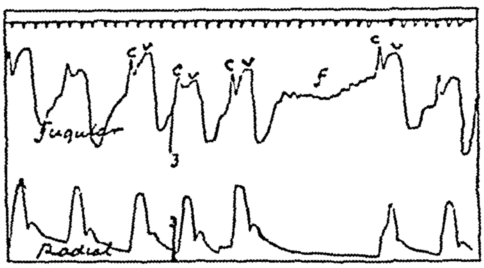


Fig 122 A tracing from a patient with auricular fibrillation, showing small fibrillary waves (*f*) in the jugular tracing (Case 44)

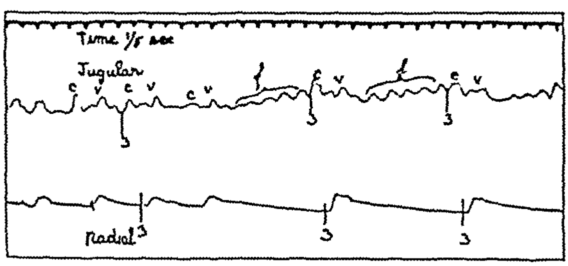


Fig 123 The jugular tracing shows coarser fibrillary waves (*f*)

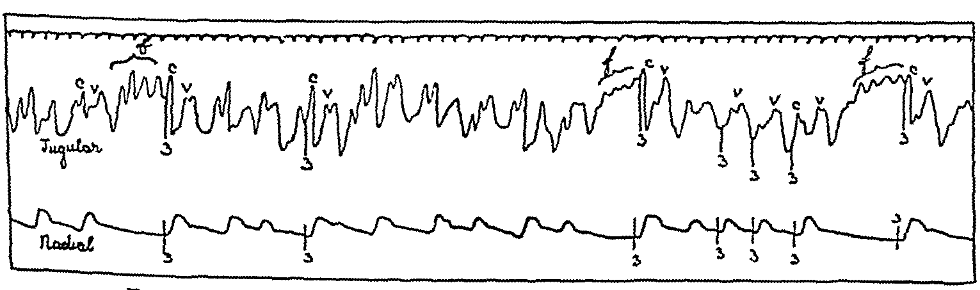


Fig 124 The jugular tracing shows fibrillary waves (*f*) of different sizes

fibrillation, he found that the arterial pulse became irregular, and the venous pulse changed from the auricular to the ventricular form Pursuing his investigations further, Lewis was able to detect in the electrocardiogram of experimentally produced fibrillation, certain oscillations during ventricular diastole, which were induced by the fibrillating auricle Examining more critically the electrocardiograms of typical cases of nodal rhythm which I sent to him, he found these oscillations also present, and demonstrated their correspondence with the small fibrillation waves I had noted in the jugular pulse

When Lewis placed these facts before me, I had no hesitation in abandoning my views, and accepting the fact that these cases owed their abnormal action to auricular fibrillation, and I now recognize that the reason those evidences of auricular activity, to which I have referred, disappear, is because the auricle ceases to act as a contracting chamber

Rothberger and Winterberg had independently, in 1909, drawn attention to the fact that in *pulsus irregularis perpetuus* the electrocardiogram corresponded to that of auricular fibrillation experimentally produced

In the investigation which I have been carrying on for so many years, I was not content merely to discover the mechanism by which the irregularities were produced, but I always kept before me the bearing these symptoms had on the patient's present and future state, and what indications they gave for treatment To this end, I made careful notes of all attendant circumstances, such as the patient's history, the size of the heart, the degree of heart failure, the response to treatment, and the future progress of the case The result is, that though I failed to recognize the nature of the altered rhythm of the heart, yet from my notes of a great number of individual patients, many of whom I had watched for a number of years, I had been able to study many of the characteristics of this group and to recognize the clinical features

Up till 1908, these observations were carried on in my work as a general practitioner Since 1909 I have reviewed the whole subject, first at the Mount Vernon Hospital, and then at the London Hospital, with the assistance of my colleagues, and we have verified the main features I had previously recognized, and extended our observations Although we can recognize many salient features of the condition, there is still a great deal of work to be done, before a full knowledge of the change in the heart's action under this new rhythm can be acquired

What is auricular fibrillation?—The term "fibrillation" is applied to a curious condition of the muscle fibres of the heart, where the individual fibres, in place of contracting in an orderly and simultaneous manner during systole, contract rapidly and independently of one another The auricle, when in a state of fibrillation, presents an entirely different aspect from what it does during its normal action

"The walls of the auricle stand in the diastolic position, systole, either complete or partial, is never accomplished, the wall, as a whole, is stationary, but careful examination of the muscle reveals an extremely active condition, it appears to be alive with movement, rapid, minute, and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface" (Lewis)

When the ventricle passes into fibrillation, the circulation is at once brought to a standstill, and MacWilliam has suggested that this is probably the cause of sudden death in the human subject When the auricles

pass into fibrillation, death does not ensue, for the fibrillation cannot pass along the bundle which connects auricle with ventricle.

Conditions inducing auricular fibrillation.—In experiment, auricular fibrillation can be produced by electrical stimulation of the auricular wall. In the human heart it is found to arise under a variety of conditions. It is probable that it is produced by altered nutrition of the muscle. Thus, I detected it in 1892 in a patient recovering from a mild attack of rheumatic fever, and there was no other evidence that the heart was affected. The attack passed off after some hours, and the youth has grown up into healthy manhood with no evident lesion of the heart. I have seen it appear in the heart in pneumonia, during the attack and during convalescence with disastrous results in both cases. Digitalis can induce it in predisposed cases. I have known it to occur intermittently in a fatal case of infective endocarditis, and Price has shown its occurrence in a fatal case of diphtheria, and G. A. Sutherland in a severe attack of rheumatic fever. Post-mortem examination in these cases showed that marked changes of an inflammatory nature had occurred in the walls of the auricle.

Effort, sometimes slight, and sometimes violent, may provoke auricular fibrillation. This occurs most frequently in the middle-aged or elderly, or in those with some old rheumatic affection. Thus, a healthy and vigorous member of our profession at the age of 50 years ran rapidly for 200 yards, and was seized with an attack which lasted for two hours. This was ten years ago, and he is still well and actively engaged in his work. In many cases, these attacks lasting for a short period are apt to recur with increasing frequency until they become permanent. While they are occurring intermittently, they are often easily provoked by effort, though they may not infrequently arise from no apparent cause. Thus, in one man under my care at the Mount Vernon Hospital, the heart would be detected beating irregularly several times a day, the irregular period lasting from half an hour to two hours. This irregularity was due to auricular fibrillation, as shown by records by the polygraph and electrocardiograph. He himself was not conscious of the altered rhythm, nor was there any recognizable cause for the onset.

We are not yet in a position to decide, with sufficient accuracy, the nature of the changes in the heart-wall which favour the occurrence of auricular fibrillation. In the hearts which I have had examined, which showed auricular fibrillation during life, there has been found in the auricle and ventricle, an increase of fibrous tissue and of nucleated cells in the muscular walls (see Cases 48, 49, and 51). In most cases, there is probably some definite change which predisposes to this condition, and it only needs an adequate stimulus to provoke it. This stimulus may be of a varied kind, for while the onset can frequently be traced to violent bodily effort, it often occurs when there is no excessive effort. At pres-

ent, we can only say that one predisposing condition is certain organic changes in the muscle wall of the auricle

Duration of auricular fibrillation—In the majority of cases when auricular fibrillation sets in, it persists for the remainder of the individual's life I have watched individual cases for over thirteen years, in whom it was constantly present (see Case 43) In many cases, it may appear for a few hours, and may never recur, or it may recur at infrequent intervals for some weeks and months, and then disappear Many cases of paroxysmal tachycardia owe the paroxysms to auricular fibrillation, and in such cases it may last for a few seconds, a day or two, a few weeks, or even months (see Case 51) As a rule, however, when it is intermittent in its appearance, the tendency to its recurrence becomes greater, till finally it becomes permanently established

Effect on the ventricles—In fibrillation of the auricle, the stimulus for contraction arises no longer in the sino-auricular node, but in the fibrillating fibres of the auricle, and is transmitted to the auriculo-ventricular node in an irregular manner It is probable that the manner in which the ventricle is affected depends on the power of the node and bundle to receive and transmit the auricular stimulations, for I found that in some of my cases the ventricular rate varied very much, sometimes being rapid and sometimes slow (see Cases 44, 51, and 54) It might be suggested that with the onset of auricular fibrillation the ventricle takes on a rhythm of its own, and indeed at one stage of my investigations I had suggested an idio-ventricular rhythm But, in experiment, if after fibrillation has been set up, the bundle is cut, the ventricular rate at once alters, and the ventricle assumes its own peculiar slow rhythm

Rate—Changes in the rate and rhythm of the ventricle, and in the size of the heart, are very common with the onset of auricular fibrillation In several cases, I have detected these changes shortly after its inception I have found the greatest difference in rate, ranging from 40 to 130 beats per minute It is but seldom that we get the opportunity of seeing auricular fibrillation start, as the patient is frequently unconscious of the change in the heart's rhythm, though some recognize the curious sensation of fluttering The patient may consult us because of the distress which may sooner or later appear, and then we usually find the rate remarkably increased, generally between 110 and 140 beats per minute and over I have met with a number of cases, in which the rate has become slower on the inception of auricular fibrillation When fibrillation has arisen as the result of the administration of digitalis, the rate has been infrequent (Case 92) If digitalis slows the heart in fibrillation through its action on the vagus and the auriculo-ventricular bundle, we might assume that the slowing occurs in consequence of some affection of the auriculo-ventricular bundle In support of this view I have observed

a case for many years, in which there was persistent increase in the interval between the auricular and ventricular systoles, and in which at one time there was partial heart-block. When the patient's auricle started to fibrillate, the ventricular rate fell from 60 to 40 beats per minute, and has continued at this rate for nine years. Cases 80 and 81 show a slow pulse-rate, but in these there was present complete heart-block. In Case 79, the heart fell suddenly to 40 beats per minute and was quite regular, the slow rate persisted for a fortnight, when it suddenly increased in rate and the auricle resumed its normal action. In this case, during its normal action, there were no evidences of heart-block.

Rhythm.—When the auricle passes into fibrillation, the ventricle usually becomes irregular in its action. The alteration is sudden, as shown not only by experiment but by clinical observations, in cases in which I have actually observed the change in the heart's action. The cessation of fibrillation can be recognized by the return to a regular rhythm, occasionally the return is accompanied by a few irregular beats, due to extra-systoles. Though many speak of this condition as the *pulsus irregularis perpetuus*, I have seen a number of cases where the rhythm of the ventricle was regular. In the majority of such cases, the rate was under fifty beats per minute. In several instances, the slow regular action has been induced by digitalis. In a case recently under observation, the rate under digitalis fell from 110 to 70 beats per minute. Prior to the administration of digitalis, the rhythm was very irregular, but when the rate fell to 70 beats per minute, it was quite regular. The patient had no jugular pulse, but by the electrocardiograph, Lewis demonstrated that auricular fibrillation was present.

The character of the irregularity as seen in the pulse is a completely disorderly one, in the sense that the interval between the beats is ever varying, two successive beats being seldom of the same length. Although, as a rule, there is a distinct relation between the size of the beats and the length of the preceding pause, the longer pauses being followed by bigger beats, not infrequently this is not so, big beats sometimes following very short pauses.

Many other conditions produce continuous irregularities, so that we have to be careful not to form our opinion on the irregularity by itself.

The size of the heart.—In the great majority of cases, a considerable enlargement of the heart follows the inception of auricular fibrillation. Though the auricles are often greatly distended, it is not possible to tell from clinical examination, how much of this enlargement is due to the auricles, and how much to the ventricles.

With the onset of fibrillation, the increase in size does not take place at once, though, in a few cases of periodic fibrillation, I have seen the heart increase greatly in size within a few hours of the onset, and the enlargement would disappear within a few hours after the cessation of

the fibrillation As a rule, little change in the size of the heart takes place at first, and if the heart is capable of maintaining an efficient circulation little increase in size may be detected for years In the majority there is an inability to do the work efficiently, so that gradually the heart's strength becomes exhausted, and an increase in its size follows With *appropriate treatment, a considerable diminution may take place, but* this is by no means constant I have been surprised at its persistence in cases of old-standing auricular fibrillation, in whom a very striking improvement of the heart's condition has taken place as the result of treatment In most cases we have carefully studied at the Mount Vernon Hospital, and at the London Hospital, we have failed to detect any decrease in the size of the heart, in patients who had suffered from extreme failure, and who had made a surprising recovery

The jugular pulse in auricular fibrillation—The size of the jugular pulse is extremely variable, in different cases, and in some individuals at different times This is in a great measure due to the amount of distension of the right side of the heart and of the great veins, and also to the rate of the heart In some slow-acting hearts, great waves can be seen extending up the neck with each contraction of the ventricle, and there is no difficulty in recognizing their nature In other hearts beating at more rapid rates, these waves are also evident, one cannot be always sure of their nature from inspection alone, but a tracing will show them to be of ventricular form When the veins are less full, and the rate frequent, it is utterly impossible to differentiate the waves that may appear in the veins Even in a graphic record some difficulty may be met with, but if the time of the waves be accurately placed in the cardiac cycle in the manner already described, as a rule the features of the tracing can be recognized In many cases, the character of the ventricular waves shows a curious difference Thus some slow beats will show a high wave at the same time as the carotid pulse (marked *c* in the tracings), and a great fall during the mid-systole of the ventricle, with a wave towards the end of systole, which ends as usual with the opening of the tricuspid valves With the more rapid beats the fall during mid-systole disappears, the characteristic wave of the ventricular venous pulse is shown The fall is due to the dragging down of the auriculo-ventricular septum during ventricular systole

Fibrillation waves—There is an additional feature, which is also of value in recognizing the presence of auricular fibrillation, and that is the presence of the small waves caused in some way by the fibrillating auricle They are not present in every case, nor always perceptible in those cases that show them When present, they are most evident during those long pauses of the ventricle which are so frequent in auricular fibrillation These fibrillation waves are of a variable size, sometimes very minute,

as in Fig 122, and sometimes very coarse, as in Fig 123 In Fig 124, they will be found to vary in size and duration at different times

The electrocardiogram in auricular fibrillation—While the normal electrocardiogram shows features similar to those in Fig 16, a great number of differences can be obtained, each due to some abnormal action of the heart So far as the electrocardiogram of auricular fibrillation is concerned, in addition to the irregular action of the ventricles, the records show some very characteristic signs, the chief being a total disappearance of the peak, P, due to the auricle The variations R and T, due to the ventricle, maintain their characteristic form Between the ventricular beats, the records may show a series of small movements, which we now recognize as being due to the fibrillating muscle of the auricular wall This absence of the auricular movements and the presence of the movements during ventricular diastole are the means, by which auricular fibrillation can be recognized in the electrocardiographic record Further, there is also the characteristic disorderly rhythm

Dr Lewis tells me, that the small movements shown in the electrocardiogram are not constant, but in every given case they come and go for no apparent reason In this they differ from the movements in auricular flutter, which are constant There appears to be some relation between these movements and the fibrillation waves found in the jugular tracing, and as these come and go, in an unaccountable way, it is probable that they are both due to the same factor

Changes in the heart's murmurs—I have already dealt with the evidences of auricular activity, obtained by graphic and electrocardiographic methods The evidence of auricular activity obtained in the clinical examination, apart from the graphic records of the jugular pulse, is limited to the murmurs of mitral stenosis We must bear in mind that stenosis of the mitral valve is a gradual process, at first not recognizable until a certain degree of narrowing has arisen This narrowing obstructs the flow of blood from auricle to ventricle, and gives rise to a murmur on the contraction of the auricle A presystolic murmur is evidence of a contracting auricle, and is usually an indication not only of an obstruction to the flow of blood from auricle to ventricle, but also of gradually progressing fibrotic changes in the valves and around the mitral orifice With progressive narrowing, the presystolic murmur becomes louder and longer, while a new murmur may appear after the second sound This diastolic murmur is due to the obstruction of the flow of blood through the mitral orifice at the end of ventricular systole It is faint and short at first, but with further narrowing of the mitral orifice it increases, till it fills up a great portion of the diastolic period of the cardiac revolution, and may run up to the presystolic murmur When this happens the diastolic period is filled up entirely by murmurs We may take it that the appearance of this diastolic mitral murmur is al-

ways an evidence, that the progressive narrowing of the mitral orifice has reached an advanced stage But the fibrotic changes that cause mitral stenosis are not limited to the valvular orifices, but are also present in the muscular walls of the heart These changes in the auricular wall predispose to auricular fibrillation, and this may arise at different stages With the onset of fibrillation, a change takes place in the character of the murmurs

If a presystolic murmur, due to the auricular systole, was present prior to the onset of the fibrillation, it at once disappears when the pulse becomes irregular If a diastolic murmur, due to mitral stenosis, has been present, it persists, because it is caused, not by the systole of the auricle, but by the inrush of blood from the auricle into the ventricle when the ventricle relaxes after its systole I wish to emphasize this change in *the character of the murmurs in mitral stenosis with auricular fibrillation*, for even those who detect the clinical symptoms of auricular fibrillation do not seem to have grasped the significance of the change in the murmurs I have carefully studied a large number of cases of mitral stenosis with fibrillation, and many of them have come to an autopsy, and in none of them have I detected a presystolic murmur of the crescendo type Where a murmur has preceded the first sound, it has filled the whole diastole when the rate was rapid When the heart's rate is rapid in auricular fibrillation, this diastolic murmur often fills up the whole space between the second and first sounds, and it may simulate, and is often taken for, a presystolic murmur due to the systole of the auricle If, however, this murmur be noticed during one of the long pauses, which occur frequently in most cases, or when the heart's rate becomes slow, it will be found that this murmur follows the second sound, but pauses some little distance before the first sound, and there is a silence immediately before the first sound in the place where the crescendo presystolic murmur due to the auricle should appear

This is brought out in the diagram in Fig 125, where A and B represent the sounds and murmurs in mitral stenosis before and after auricular fibrillation In B it will be seen that there is no presystolic crescendo murmur, and that the diastolic murmur fills up the whole of the space between the second and first heart sounds, when the interval is short (x,x), but when the interval is long (y,y) the diastolic murmur does not reach the first sound, and there is a silence before it

From these considerations, we can, in the great majority of cases, conclude that auricular fibrillation is present when there is a diastolic mitral murmur without a presystolic murmur As a rule, the irregular action of the heart is also suggestive, but in many cases of mitral stenosis with auricular fibrillation, when the patient is under digitalis, the heart becomes slow and almost, or even quite, regular

The explanation given here occurred to me in 1897, as an outcome of the study of the features of Case 48. Since that time I have continued the observations and verified it repeatedly, but I have found the greatest difficulty in convincing physicians of the clinical facts. The murmur filling up the interval between the second and first sound is invariably looked upon as presystolic, while the long diastolic murmur present when the heart is slow (see Fig. 192*), is not infrequently taken to be aortic; on sundry occasions I have heard physicians express surprise that at the post-mortem examination there has been no aortic lesion, but mitral stenosis in cases with the long diastolic murmur. Quite recently Dr. Lewis has taken records, by means of the electro-phonograph, of cases of auricular fibrillation, and his results have confirmed the explanation given above.

Auricular fibrillation and digitalis.—Not the least in importance of the discoveries resulting from the recognition of auricular fibrillation as a clinical entity, is the light that is thrown upon the action of drugs of

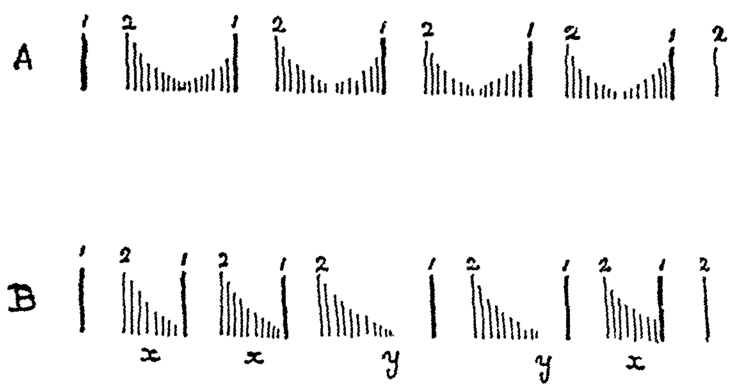


FIG. 125.—Diagram illustrating the change in the murmurs in mitral stenosis when auricular fibrillation occurs. The perpendicular lines 1 and 2 represent the first and second sounds of the heart, and the shading between the second and first sounds represents the diastolic and presystolic murmurs with a regular rhythm *(A)*. In B auricular fibrillation causes the rhythm to be irregular, and when the diastolic period is short as at *x, x, x,* the diastolic murmur fills up the whole interval. When, however, the diastolic period is lengthened as at *y, y,* the diastolic murmur does not fill up the whole period, and there is then a silence before the first sound when the presystolic murmur was heard before fibrillation set in.

the digitalis group. I can only here refer, briefly, to a few points which I have been able to elucidate. I think every one who has carefully studied the description usually given of the effects of digitalis on the human heart, cannot but be struck with the absence of agreement among the different writers, as to the manner of its action, its dosage, and the best preparation. In some instances, some peculiar reaction which the observer may have noted is looked upon as the characteristic effect of digitalis but the mechanism of this peculiar reaction has not been understood. It is a good many years since I was struck with the varied reactions, which I obtained from the use of digitalis. I collected a great number of cases; in some I got a definite reaction on the heart, while in others no reaction was obtained. When I separated these into groups,

*Appears in chapter beyond that reprinted in this book.—F. A. W., 1940.

I saw that the probable reasons for the varied reactions in the human heart were, that digitalis gives a reaction according to the nature of the lesions from which the heart is suffering It will be observed that if this is found to be correct, we can at once understand how the physiologist and experimental pharmacologist have missed the most important effects of digitalis, for, so far as the heart in experimental work is concerned, they cannot reproduce the conditions under which the physician has to employ the drug

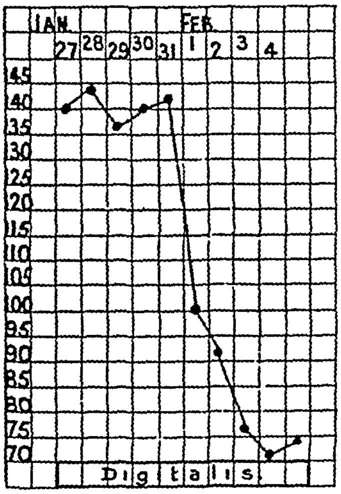


FIG 126 Chart showing a typical reaction of digitalis in a case of auricular fibrillation with severe heart failure The administration of the tincture of digitalis began on January 27, in doses of 15 minims four times daily

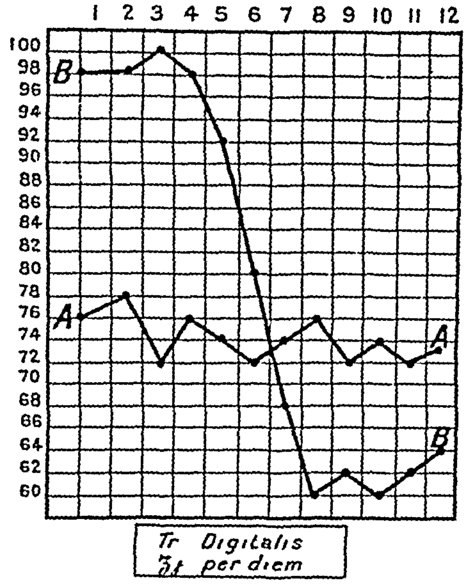


FIG 127 Chart showing the effects of similar doses of tincture of digitalis on six cases of mitral stenosis with the normal rhythm (*A*) and six cases of mitral stenosis with auricular fibrillation (*B*) The figures at the side represent heart beats, those at the top, days In each case the digitalis was continued till the heart became slowed, or until nausea or vomiting occurred The average quantity before an effect was produced was 7 drachms (1 drachm per diem) This had little or no effect on the heart rate in the patients with the normal rhythm (*A*), while there was a rapid decrease in the rate in the patients with auricular fibrillation (*B*)

It is not only in auricular fibrillation that digitalis acts beneficially, for there are many other conditions which benefit by it, but cases of fibrillation stand apart from all others in regard to their response to this drug All cases of auricular fibrillation are not responsive, for there are factors which render certain hearts unsusceptible, as the presence of fever or extensive fibrous degeneration It is in certain cases, where there is a fair amount of healthy muscle that its almost specific action is seen It is some ten years, since I realized this peculiar response to digitalis When I was appointed to the Mount Vernon Hospital and London Hospital, I seized the opportunity to start a series of observations under conditions, which permitted a degree of accuracy unattainable in

private practice In these observations, the same drug and the same dose were given to patients with and without auricular fibrillation With only rare exceptions, all the cases that showed a marked effect upon the heart were cases of auricular fibrillation, for, although the other cases might exhibit some benefit from the use of the drug, they never showed the same tendency to slowing of the heart's rate

The slowing effects of digitalis are shown in a very striking manner in those cases of auricular fibrillation, where heart failure set in with a great increase of rate of the heart The chart (Fig 126) is a good illustration of these types The patient from whom the chart was obtained, suffered from extreme heart failure, and the rate of the heart was 140 per minute and very irregular She was given one drachm of digitalis per day, and after five days the pulse-rate fell in the manner shown in the chart At the same time, there was a remarkable improvement in the patient's general condition

The difference in the reaction of hearts affected with auricular fibrillation and those with the normal rhythm is well brought out by the chart in Fig 127, where the average heart-rate from six cases of mitral stenosis with auricular fibrillation is compared with six cases of mitral stenosis with the normal rhythm The record begins with the rate on the day previous to administration of the drug, and it will be seen that the rate in the cases with auricular fibrillation is greater than the rate in cases with the normal rhythm This, I may remark in passing, is a point of some interest, for cases of heart failure with mitral stenosis with the normal rhythm, rarely, have as rapid a pulse as those with mitral stenosis with auricular fibrillation

In each case the tincture of digitalis was given, one drachm per day, and was continued till nausea or vomiting ensued

Digitalis in some hearts induces auricular fibrillation When this occurs, the rate of the ventricle becomes greatly decreased (see Case 92)

Effect on the heart's efficiency—A great many patients in whom auricular fibrillation had occurred, suffered from lesions of the heart, which impaired its efficiency In all these, the occurrence of fibrillation at once increased the impairment, and the symptoms of heart failure became intensified In others, where there had only been a slight impairment of the cardiac efficiency, the onset of auricular fibrillation speedily provoked symptoms of extreme heart failure, while in others little difference could be detected In a few cases, the onset of auricular fibrillation has only slightly embarrassed the heart in its work

To a great extent the symptoms of heart failure arise most markedly in those in whom the change in the auricular action has affected the ventricle, particularly in increasing its rate In a few cases, marked limitation of the heart's powers of response to effort has followed, with relatively slow acting hearts

The symptoms of heart failure commonly produced are of the same kind as arise in heart failure from other causes, for example, shortness of breath on exertion and consciousness of the heart's action, particularly when some effort is made. With increase in the failure, oedema of the legs and lungs sets in. The face becomes livid, the patient cannot lie flat in bed, but has to be propped up. The liver becomes enlarged, while the veins in the neck may become engorged, and the pulsation in them becomes extremely marked. With these changes, dilatation of the heart and great increase in the rate may be detected. As a rule, the onset of these symptoms is slow and gradual, but occasionally they may set in with great rapidity, they quickly disappear if the heart reverts to its normal rhythm.

Cause of heart failure in auricular fibrillation.—However it may be brought about, the onset of fibrillation embarrasses the ventricle in its work, and *in all probability the degree of heart insufficiency that results* depends upon the extent of concomitant damage of the ventricles, and the amount of embarrassment caused by any valve lesion that may be present. There can be little doubt that the orderly action of the auricle in regulating the supply of blood to the ventricle, and in stimulating it in a normal manner, results in a more efficient action of the ventricle than the variable and irregular stimulation to contraction. When the ventricle is rapidly and irregularly stimulated to contraction, there results a gradual exhaustion of the strength of the ventricle, and evidences of heart failure supervene. In cases that have drifted and died, where I have had a post-mortem examination, there has been found extensive fibrous degeneration of the heart muscle (Cases 49 and 51). On the other hand, I have seen so many individuals with auricular fibrillation who have led strenuous lives, engaged in hard manual labour, that I infer that in them the muscle of the ventricle had not been seriously damaged (Case 44).

Clinical characteristics. *The patient's history.*—The conditions which induce auricular fibrillation vary, but there are two classes among whom it is very frequent—namely, patients with a heart affection following upon rheumatic fever (frequently associated with mitral stenosis), and elderly patients with fibrous degenerative changes, usually spoken of as "senile." It may be found in the young and middle-aged, with no history of infection. It is customary in rheumatic hearts to take into account only the valvular changes, while as a matter of fact the really serious element is the slow, insidious change in the muscle that has started during the attack of rheumatic fever, and which finally provokes fibrillation of the auricle.

The patient's sensations.—Many people become conscious of the heart's action when it departs from its normal rhythm. Thus, extra-systoles are sometimes recognized by the individual's consciousness of the long pause,

or of the big beat which follows the pause The patients liable to paroxysmal tachycardia, are conscious of the attack by the feeling of a gentle fluttering sensation in the chest When the attacks of tachycardia are due to auricular fibrillation, this fluttering is also present, but usually it is not a continuous fluttering sensation, but is interrupted by thumping sensations, due to the occasional occurrence of bigger beats This consciousness of the heart's action is frequent in cases of auricular fibrillation, the patients being conscious of the fluttering and irregular action of the heart In many cases, where the heart does its work efficiently, these sensations are not perceived, unless the heart is submitted to over-exertion, or when it begins to fail

Character of the pulse—The symptom by which the clinical observer can most readily recognize this condition, is by the character of the pulse, the rhythm usually being irregular, and the irregularity of a very disorderly kind Irregularities, apart from those due to auricular fibrillation, usually have a distinctive character, as the irregularity in the heart of the young, where variations in rate coincide with phases of respiration, as the intermittent pulse, or the irregular heart due to extra-systoles, the irregularity breaking in on an otherwise regular rhythm, unless it occurs alternating with a normal pulse-beat In auricular fibrillation, as a rule, the pauses between the beats are continuously changing and two succeeding beats are rarely of the same strength, or the pauses between the beats of the same duration The character of the irregularities will be better recognized from the radial tracings, as shown in Figs 122, 123, and 124

Symptoms of heart failure—The sign which usually calls attention to auricular fibrillation is the patient's consciousness of his limitation The signs of this limitation, however, are not peculiar to or characteristic of auricular fibrillation, but are common to heart failure induced by other conditions These signs of heart failure may range from a slight breathlessness on exertion, to dyspnoea of the most severe kind, accompanied by dropsy, enlarged liver, and the symptoms associated with extreme heart failure

As a rule, the onset of symptoms of heart failure is slow and gradual, due in a measure to the individual's persisting in living in his usual manner, though the heart is hampered by the abnormal action On the other hand, the onset of heart failure may be very rapid Within a few hours of the occurrence of auricular fibrillation, the distress of the patient may be very severe, the countenance dusky, the heart dilated, orthopnoea, and a feeling of distress is experienced I have seen these phenomena arise rapidly in cases, where the fibrillation occurred intermittently, and the relief experienced by the patient, when the heart resumed the normal rhythm, was as remarkable and striking as the onset of the suffering At once the patient knows that the heart's action has altered, and he breathes easier, and the feeling of distress disappears

Within a few hours, the heart and liver have become reduced in size, the lividity of the face has gone, and the tenderness of the chest-wall and over the region of the liver speedily disappears.

In many cases, the persistence of heart failure is accompanied by wasting, the patient sometimes losing a good deal of weight in a few months. Accompanying this, there is usually a certain amount of flushing of the cheeks, usually of a dusky colour, and occasionally a slight sallow tinge of jaundice. These symptoms with an enlarged liver may be mistaken for sarcoma of the liver.

Auricular fibrillation and angina pectoris.—Amongst the more common signs of heart failure, there is one which I have but rarely seen—namely, definite attacks of angina pectoris. As I have already stated, pain and hyperalgesia are not uncommon in the heart failure associated with auricular fibrillation, while I have met with typical attacks of angina pectoris in only a few cases. In quite a number of cases where the patient had angina pectoris, the attacks ceased with the onset of the fibrillation. Needless to say, the onset of auricular fibrillation in those cases induced such embarrassment to the heart's action, that the patients all drifted, and only lived a few months after its onset. The onset of fibrillation may be associated with angina pectoris (Case 50).

Prognosis.—We must bear in mind that auricular fibrillation is in reality a symptom of some myocardial change, and that, to be logical, we should only consider it from the point of view of a myocardial affection. We are at present so ignorant of myocardial disease, that we are forced to put one symptom forward as if it were in itself a disease. Illogical as this seems, it has its use, for the occurrence of auricular fibrillation induces such a profound change in the heart's action, affecting its efficiency, reacting on the ventricle, and modifying its behaviour to drugs that we are compelled to look upon it as a condition apart. Considering the variety of conditions that induce auricular fibrillation, it is difficult to state briefly its prognostic significance. In referring to the pathological lesions associated with it, I showed that they were of a very diverse kind in nature and in degree. It is in all probability the extent of these pathological changes which determines the prognosis of auricular fibrillation, and an attempt should be made to estimate their extent. If we look upon the inception of the new rhythm as in itself embarrassing the heart in its work, then the maintenance of an efficient circulation depends on whether the heart is able to do its work, when hampered by the new rhythm. That this is the fundamental question will be recognized, when we study the effect of fibrillation in certain individuals. I have repeatedly seen individuals in whom fibrillation occurred for a short time, and in whom heart failure set in with an extraordinary rapidity, the patient becoming breathless, having to sit up in bed, the face becoming livid, the heart dilated, and the liver swelling, within a few hours.

after the onset of fibrillation. With the restoration of the normal rhythm, these symptoms quickly disappeared. When fibrillation became permanent in such individuals, the signs of heart failure persisted sometimes in spite of all treatment, till death supervened in a few weeks or a few months (Cases 51, 52, and 53).

On the other hand, I have repeatedly seen fibrillation set in, and the individual be altogether unconscious of its presence. These cases may go on for years with little inconvenience, but the majority after some years gradually show signs of a limitation of the field of cardiac response, and their future depends upon how they respond to treatment, and on their ability to diminish the amount of their bodily work, and to live within the limits of the heart's strength (Cases 43, 44 and 45).

Much more frequently there is a considerable limitation of the heart's power of response to effort, and if the usual life of the individual be pursued, without appropriate treatment, there is a great tendency for the heart gradually to fail. There is no doubt that the onset of fibrillation can lead directly to a fatal termination, or, rather, can be associated with conditions that lead to death. Thus, one of my patients died suddenly a few days after the inception of auricular fibrillation. Another one fell down dead six months after its inception. I have seen a number of other patients die suddenly, who had auricular fibrillation, but some of these suffered from a considerable degree of heart failure. It has appeared to me probable that in these cases the ventricle has passed into fibrillation, as MacWilliam suggested. This view is probable also from the fact, that the histological changes in the ventricle were similar to those in the auricle in some of the cases of sudden death.

The usual mode of death in auricular fibrillation is a steady advance of the heart failure, as shown by the breathlessness on exertion, orthopnoea, dropsy, and enlargement of the liver, etc., sometimes with an absolute failure of response to all forms of treatment. Thus, I have seen death ensue in this manner a few weeks after the inception of fibrillation (Case 52), and others have drifted on for a few months (Cases 50 and 51), while some have led a somewhat chequered career for a number of years, seldom fit for much bodily exercise.

In giving a prognosis in cases of auricular fibrillation, it is necessary to appreciate a good many other things, besides the mere presence of the fibrillation. It is necessary to form an opinion of the extent of the changes that have led up to the fibrillation, and in many cases to find out how long these changes have been going on, as, for instance, the date of an attack of rheumatic fever, and how the patient comported himself before the onset of fibrillation, if, for instance, he was liable to attacks of heart failure, in which case such attacks point to a tendency to exhaustion which may be aggravated by the fibrillation. Amongst valvular lesions, cases with affections of the aortic valve are usually

seriously embarrassed, particularly in aortic regurgitation, when, prior to the onset of fibrillation, there had been evidences of failure The character of the murmurs present in mitral stenosis will shed light upon the progress of the disease as already described When auricular fibrillation sets in, it is necessary to observe the accompanying changes in the heart, and the way in which it maintains the circulation Thus, an increase in the size of the heart, or a rate over 120 beats per minute, usually leads to a speedy exhaustion of the heart's strength I have occasionally met with an individual with a heart-rate of 100 and 120 per minute, with no increase in the size of the heart, who suffered little inconvenience, but as a rule any rate over 90 beats per minute tends to induce dilatation and consequent exhaustion On the other hand, when there is little increase in rate, or even when the rate is somewhat slower than normal and the response to effort good, the prognosis is usually very favourable

In those with symptoms of manifest heart failure, prognosis depends to a great extent on the way in which they respond to treatment I have already described the action of drugs of the digitalis group in patients with fibrillation, and I will show later, more fully, the action of other drugs in these cases, and the response of the heart to them, and what an important bearing it has upon prognosis

I have already said that there are a great many individuals with fibrillation who lead useful and energetic lives, and whose capacity for work is little, if at all, impaired by the new rhythm In such the prognosis is distinctly good

There are, however, so many exceptions to these details, that a clearer insight may be gained, by looking at each case from a broader standpoint, of how the heart responds to effort, not, however, ignoring the details, but giving them their due consideration It is in estimating this reserve power that we get the most valuable information It may be taken for granted that if distress is induced by exertion, so long as the exertion is persisted in, it will ultimately lead to serious heart failure On the other hand, when individuals with fibrillation are able to undertake the work equal to that done by a perfectly healthy man, there is proof of such a degree of healthy heart muscle and freedom from valvular or muscular embarrassment, that a good prognosis can be given

We meet, however, with such varying degrees of exhaustion, that an estimate must be acquired of the amount, though it is impossible to describe with accuracy what that amount may be Even with distinct limitation of the heart's power, as shown by the response to effort, the prognosis may still be favourable, so long as the patient lives within the limits of his powers, avoiding such efforts as cause him distress or exhaustion

In transient attacks of auricular fibrillation, the attacks usually tend to become more frequent, until auricular fibrillation becomes permanently established. The prognosis of such cases depends on the way the circulation is maintained, this to be estimated in the manner already described. Transient attacks may appear for a short period and then disappear entirely. In two of my cases, I detected a transient attack of auricular fibrillation twenty years ago, and the patients still lead vigorous and active lives. From the recognition of such cases, we can conclude that auricular fibrillation is not of necessity a sign of extreme damage.

A most valuable aid in prognosis may be found in observing how the patient responds to treatment. In sudden attacks of severe heart failure, when the heart's rate is over 120 per minute, it will be well to suspend judgment, until the reaction to digitalis is found out. Many such cases respond speedily to digitalis, and with the resultant decrease in the heart's rate a remarkable degree of recovery may ensue, so that the patients may be able to undertake laborious work, so long as the rate is kept down by the digitalis. This would seem to imply that the exhaustion is mainly brought about by the ventricle being stimulated to too great an activity, and that the slowing enables the ventricle to get more rest, and so regain a measure of strength. From this result, we can also gather that the ventricular muscle must be fairly healthy, and we can estimate, within certain limits, the amount of healthy muscle by the degree of recovery.

Treatment *General*.—When any individual with heart failure presents himself for treatment, it may be taken for granted that the individual has been undergoing a greater amount of exertion than the heart has been capable of performing without undue exhaustion. Hence the exhaustion of the heart's strength has been brought about in the first place by overwork. It may be that the amount of work has been small, as measured by what a healthy heart can perform, but when a heart is hampered by an inherent defect, such as auricular fibrillation, and the organic changes in the valves and muscle so commonly associated with this condition, the heart may be capable of a very limited amount of effort. With this conception of the cause of heart failure, the first and obvious course to pursue is to ease the heart of its work. In doing this much discrimination is required, and a thorough inquiry into the patient's mode of life has to be made in order to find out what circumstances, such as overwork, sleeplessness, digestive trouble, pain or work, may have provoked or aggravated the heart failure. These have to be attended to in every case, and relief may be at once afforded with the removal of the disturbing or exhausting cause.

The use of digitalis.—While it is important to attend to such circumstances in heart failure with auricular fibrillation, as in all other forms, there are circumstances in cases of heart failure with fibrillation which when appreciated help greatly, not only in the restoration of the heart's

strength, but in the prevention of heart failure. I have already dealt with the reaction of hearts affected with auricular fibrillation to digitalis from a physiological standpoint; it is in treatment of auricular fibrillation that we find the great value of this drug, and I cannot speak too highly of its therapeutic action.

It is seldom that I have been able to say that I have saved a patient from immediate peril by the use of drugs, but this I can say with confidence, that I have repeatedly seen patients in evident peril of death removed rapidly from danger, and restored to a condition of comparative health, and fit for work by the judicious use of digitalis. The manner of its application needs, however, very careful attention, for it is a drug that needs to be applied on certain definite lines, if full benefit is to be obtained from its action. I think it necessary to insist upon this point, for the somewhat "rule-of-thumb" methods of its use, so generally employed, fail to get the full amount of benefit which this drug is capable of bestowing.

To understand the action of digitalis, it is necessary to appreciate the manner in which heart failure progresses in cases of auricular fibrillation, and the way it is controlled by digitalis. It may be taken for granted that when a patient with auricular fibrillation has a pulse-rate, or, to be more accurate, a ventricular rate, of 90 beats per minute and over, he will in course of time gradually lose strength, his heart will become more feeble, and the evidences of heart failure will become more severe. This process may be very gradual, but it is very sure. On the other hand, heart failure may set in rapidly, more especially when the heart's rate rises to 120 and 140 and over. The severity of the failure, however it is brought about, compels the patient to seek rest, and we generally find such patients in bed, sitting up and breathing in a laboured fashion, with considerable distress, the heart usually dilated and the face of a bluish tinge, and possibly with dropsy and pulsation of the liver. In all such cases the prompt administration of digitalis is urgently called for, and, if given in sufficient doses, relief may be obtained in a few days, the relief being accompanied by a remarkable slowing of the pulse-rate. When this is accomplished, or when there are other signs of a sufficiency, the digitalis should be stopped for a few days, and resumed in small doses when the rate begins to increase. The rate of the pulse should be watched, and the quantity sought for which keeps the heart about 70 beats per minute. It is seldom advisable to keep the rate under 50 beats per minute, although in some cases the patient feels fittest when it is at a rate of about 50 beats per minute. In this, we must be guided by the patient's sensations, and the manner in which he responds to effort.

Even when patients suffer from only a moderate degree of heart failure, and are able to go about, it is well to place them under the influence of this drug if the pulse-rate is over 90 beats per minute, and in some

cases if it is over 80 per minute My usual procedure in such cases is to attend to any circumstance that may aggravate the heart failure, and then to give the patient digitalis until the pulse-rate is reduced If the failure is of some severity, I put him to bed until the proper effect is obtained, but where it is less in degree I permit him to go about his affairs

In all cases where the heart has been sufficiently reduced in rate, I find out the quantity of the drug that is necessary to keep the heart at the rate, at which it can perform its work with the greatest efficiency In doing this, the patient's sensations are of the greatest help, whether he is confined to bed or attending to his affairs He readily appreciates the change in his response to effort, and some such symptom as a disagreeable action of the heart or breathlessness, can be employed as an indication that the heart's strength is being exhausted Once the patient understands the meaning of these sensations, he is generally quick to perceive what digitalis does for him, and its administration can usually be left quite safely in his hands On such lines, I have seen many people lead useful lives for long periods of years with no bad effects, except when they have not taken the drug in sufficient quantities to keep the heart at the required rate

The foregoing line of treatment is applicable chiefly to cases in whom auricular fibrillation has arisen recently, or where the heart failure is of recent date In more advanced cases, when the condition has induced from time to time periods of heart failure, and there has appeared the change that accompanies chronic heart disease, such as persistent shortness of breath, enlarged liver, and dropsy more or less continuous, the persistent use of digitalis may still tend to restore a measure of strength to the heart and give relief, enabling the individual to lead a useful life, though at a lower level, for an indefinite period

In the search for an appropriate line of treatment in old-standing cases, I have used many methods and many drugs, often with little or no benefit, but in a certain proportion of apparently hopeless cases I have seen extraordinarily good results following the use of digitalis, pushed until a reaction was obtained, and then stopped for a time, and again resumed, time after time Not infrequently, after it has seemed useless to continue the drug, I have seen the individual acquire such an amount of strength as would scarcely have been anticipated

As the conditions preceding auricular fibrillation and producing it are all due to changes in the heart muscle of a slowly progressive nature, it is easy to recognize that the heart's strength cannot always be restored, and that as the amount of efficient muscle becomes reduced, a period is reached when no method of treatment is of avail

Method of administration —A great diversity of opinion is to be found in regard to the form in which digitalis should be given, and also in regard to the dosage So far as I have worked out the subject in regard

to auricular fibrillation, I see no reason for giving the preference to any particular preparation The best and most assured way, in cases of marked failure, is steadily to push the drug, whichever form be employed, until a reaction is observed Usually the digestive system is the first affected, loss of appetite, nausea, vomiting, or diarrhoea being set up, the patient usually feeling ill and miserable If the digitalis is effective on the heart, as a rule a marked slowing of the pulse is found at the same time, or even before any digestive disturbances arise In some cases, a slowing of the pulse is the first sign of a sufficiency When this stage is reached, I always stop the administration of the drug for a few days In a day or two, patients feel remarkably well and bright, and if nausea was present, it disappears The heart-rate is carefully observed, and when the rate shows signs of increasing, half-doses of the drug should be given, and the dose increased or diminished according to the manner in which it affects the rate, the object in view being to give just the amount which enables the heart to carry on its work with the greatest efficiency As I have previously stated, the patient himself by his own sensations speedily acquires the knowledge of how much of the drug is needed, and by attending to his own experiences, he will soon find out the smallest dose which is needed to give the best results

A good deal of my work has been done with the tincture of digitalis, and I may say that I have used this preparation for over thirty years, and have never yet come across an ineffective preparation, my standard being the reaction in susceptible individuals Professor Cushny has tested experimentally a number of samples from the Mount Vernon and London Hospitals, and has found each sample effective

The quantity I usually start with, where the failure is marked, is one drachm of the tincture per day, in doses of fifteen to twenty minims This is steadily pushed until a reaction is obtained, then it is stopped and employed in the manner already described Usually a reaction is obtained within a week, sometimes in a few days Where there is great distress and more urgency, I give as much as two drachms of the tincture daily, and then get a reaction in two or three days

I have frequently used Nativelle's digitalin granules, and find them also very efficacious I have found that one of these granules is equal to fifteen minims of the tincture

Other drugs, such as strophanthus and squills, have the same effect as the digitalis, and in some cases they may cause less digestive disturbance, but in the majority of cases I have found that when the digitalis is ineffective, so also are these drugs In many cases, the effects of digitalis are less disagreeable than these other drugs

In some urgent cases, it may be necessary to produce a reaction more speedily, though I have rarely failed to get a reaction in good time by

digitalis by the mouth. In order to obtain a speedy reaction, strophanthin or strophanthone may be injected into the veins. In a series of observations which have been carried out at the Mount Vernon Hospital and at the London Hospital, it has been found that in auricular fibrillation with a pulse-rate of over 140 per minute, intravenous injections of strophanthin (1/250 gr.) can reduce the rate and give relief in five or eight hours, but I am of opinion that it is only in very exceptional and urgent cases that this method is required.

Danger in the administration of digitalis.—For a long while, I was at a loss to understand the warning of authorities as to the danger of sudden death from administration of digitalis. Of recent years I have obtained an inkling into the cause of sudden death. I have been shown tracings of the slow pulse with characteristic coupled beats that occur under digitalis with auricular fibrillation, and have been informed that the patient died suddenly. On inquiry, it was found that, notwithstanding the evidences of a sufficiency, the drug had been continued in large doses. I was once asked to see a man who was said to be dying from heart failure. He had to sit up in bed and breathed heavily, his face was livid. He had dropsy, an enlarged liver, and a large and irregular heart beating at the rate of 130 to 140 per minute (auricular fibrillation). I told his doctor to push the digitalis till he showed evidences of a sufficiency, either by the slowing of the heart or nausea, and then to stop it. After five days he telephoned me that the patient was wonderfully free from distress, could lie flat, was a good colour, and the dropsy had almost gone, the pulse-rate being between 70 and 80. I told him to stop the digitalis for a few days, and if the pulse then increased to give smaller doses, and find out the exact quantity which kept the rate about 80. Three days later, he telephoned to say that the patient had been going on well, but that morning, during the doctor's visit, the patient fell back and died. I asked the doctor if he had stopped the digitalis and he replied in the negative, saying that, as it had done him such a lot of good, he had continued it, in spite of my directions.

On making inquiries in a few other cases where I had heard of sudden death, I had no difficulty in recognizing that they were cases of auricular fibrillation, in which digitalis had been pushed after it had affected the heart. Seeing that I have been following this line of treatment by pushing the drug till I get evidences of its action, then stopping it and resuming it later, for over fifteen years and have never had a sudden death, I am disposed to think that just as we recognize the danger of pushing chloroform beyond a certain stage, so there is danger when digitalis is pushed too far, whereas if the indications I have given are followed, such a catastrophe as death need not occur.

SIR JAMES MACKENZIE'S HEART

By

DAVID WATERSTON

With an Account of His Clinical History by JAMES ORR
And Notes on the Pathological Histology by D F CAPPELL

From the James Mackenzie Institute, St Andrews, and the Department of Anatomy, University of St Andrews

The following description of Sir James Mackenzie's heart has been prepared in accordance with his desire, expressed to myself and to other friends, that after his death his heart should be examined to ascertain what information it furnished upon the symptoms that he had experienced He died in London on January 25,† 1925, aged 72 Some weeks before his death he told Dr John Parkinson that he wished him to make a post-mortem examination This request was confirmed, after his death, by his brother, Sir William Mackenzie, now Lord Amulree The examination was performed some fourteen hours after his death, by Dr John Parkinson, assisted by Dr J W Linnell The heart was removed and subsequently sent to me at St Andrews for further examination Dr Parkinson noted that nothing abnormal was found in the pericardium

In order to correlate the clinical symptoms with the pathological condition, it has been necessary to compile an account of Sir James Mackenzie's illness This has not been easy, for, like so many other doctors, he had not been under the care and observation of a medical man from the commencement of his illness His own case is referred to both in his book on angina pectoris (Case No 28) and in that on diseases of the heart, as well as in the Reports of the St Andrews Institute

While he was in London, from 1908 to 1918, he mentioned to Sir Thomas Lewis that his anginal history began suddenly While he was in St Andrews, from 1918 to 1924, engaged in founding the Institute for Clinical Investigation which bears his name, he was on many occasions examined by Dr James Orr, and discussed with him his condition Dr Orr also saw him during several of the attacks of angina, from which he suffered with increasing severity as the years went on After his re-

*Brit Heart J 1 237-248 1939

†A discrepancy occurs regarding the date of death in this report the correct date apparently was January 26 —F A W

turn to London in 1924 he was not under the care of any medical man until very shortly before his death, when he was seen by Dr Parkinson and Dr C M Anderson

Dr Orr, who had seen him during the whole of his stay in St Andrews, has written the account of the clinical history which follows My colleague, Professor D F Cappell, undertook the histological examination of the blood vessels and heart muscle, and his notes on them are included in the description

CLINICAL HISTORY AT ST ANDREWS

The medical life history of Sir James Mackenzie is the story of the onset and gradual progress of angina pectoris from sclerosis of the coronary arteries He had a mild attack of typhoid fever in 1880 and an occasional attack of renal colic in his later years, but suffered from no other illness

With the exception of a tendency to extrasystoles commencing at the age of forty, the first evidence of real cardiac involvement was in 1901, at the age of forty-seven This was a heart attack with irregularity of the pulse, which occurred after running 300-400 yards In his own description of this attack (Mackenzie, 1925) he notes that he "was conscious of a slight fluttering sensation, but suffered no distress of any kind" The pulse rate during this attack, which lasted two hours, was 90 per minute A tracing, taken by himself, showed auricular fibrillation During the next four or five years several attacks of this kind occurred, mostly after a full meal or when walking up a hill, they lasted from ten minutes to half an hour and never caused any distress or limitation in his powers of walking

The earliest symptom of limitation of effort was noticed by himself in 1907, and was represented by a slight feeling of constriction, hardly amounting to pain, in the upper part of the chest on severe continued exertion, and which soon ceased with rest There were long periods when it was not experienced at all For two years he was conscious of slight pain on effort under certain conditions, such as walking after a full meal or on a cold day This pain he described as preceded by a sense of tightness or constriction, such as used to pull him up when running a race in boyhood

In 1908, at the age of 55, Mackenzie experienced his first severe attack of cardiac pain It occurred at night when resting, and followed a period of dining out at frequent intervals The pain was across the chest and down the left arm, it lasted two hours and varied in severity Mackenzie further notes that in this attack "he could not be still but had to move about" After 10 grains of veronal sleep was obtained, and next day he was quite well and free from pain, though walking in the cold or

after a meal still produced discomfort of an anginal type This gradually became more noticeable, and by 1911 there was definite limitation of effort, though pain could be avoided by careful regulation of effort From this time until the end of his life a somewhat anomalous symptom was present, to which he often referred, namely, that while a sustained effort produced pain, a sudden effort produced breathlessness without pain

Mackenzie came to St Andrews in 1918, and at that time was able to walk at any pace from his home to the Cottage Hospital without discomfort, a distance of two miles In 1919 when I first examined him, he was still able to do this and could play a round of golf regularly The heart was then ¼ inch external to the mid-clavicular line, the sounds were closed and well spaced, and except for an occasional extrasystole, the rhythm was regular Blood pressure was 156/92 mm At this time he was also affected with intermittent claudication on continuous walking He had first noticed this ten years previously after a rapid four-mile walk, but in 1919 it was evident after a short half-mile walk The posterior tibial pulse was well felt on both sides During the next five years this symptom was much less pronounced owing to the fact that pain in the chest occurred in response to a smaller effort than was necessary to produce claudication

In 1922 limitation of effort prevented his playing golf, and even walking became difficult though by careful regulation of effort, severe pain was, in the main, avoided A few very severe attacks occurred, like that in 1908, while resting The most severe of all, in 1923, happened while he was sitting in his study in the afternoon, and lasted nearly an hour, it was little influenced by nitroglycerine and was followed by extreme exhaustion In August 1924 Mackenzie returned to London, and by this time only the gentlest of exercise was possible Death followed a very severe and prolonged anginal attack in January 1925 As has been already mentioned, Dr John Parkinson saw him shortly before his death, and has supplied the following note

"On January 24 and 25, 1925, he suffered severe and prolonged attacks of anginal pain, and Dr C M Anderson was called during the night At 4 30 A M on January 25 he had morphine subcutaneously, gr ½, and chloroform inhalation for about an hour It was necessary to repeat both at 8 20 P M on that day At this time the pulse was 100 and regular and there was Cheyne-Stokes breathing I did not myself see him until 10 30 P M that night (January 25), and he was then asleep About 1 A M on January 26 he awoke free from pain and perfectly conscious and composed He conversed cheerfully with Lady Mackenzie and me for a few minutes and then said he felt sleepy and soon he slept At 4 A M his breathing changed and became irregular with long pauses, and a few minutes later the pulse stopped There were no indications of pain at the end"

The heart was uniformly enlarged Its weight was 18 ounces (510 g)

.

The left ventricle was a large and thick walled chamber The muscular wall, for the most part thick and firm, was 27 mm in thickness near the base At the apex, as usual, it was thin, and only 3 mm in diameter In colour it was somewhat pale In its substance were several small whitish patches of fibrous tissue, in size from a pin's head upwards In the anterior wall, 30 mm above the apex, there was a patch of fibrous tissue 8 by 3 mm and another patch of similar structure and size lay in the substance of the posterior wall, about midway between apex and base At the apex there was a small recent haemorrhagic infarction involving the deeper part of the muscle wall, covered by a nodular reddish brown mass of clot the size of a cherry stone which projected into the cavity of the ventricle Section through this and the adjacent wall showed that the nodular tissue extended into the substance of the muscular wall, which was here reduced to a narrow margin 3 mm thick

.

The aorta had been divided 6 to 7 cm from its root At the point of division the lumen was cylindrical, measuring 33-34 mm The ascending aorta showed a bulging to the right side (the bulb of the aorta) by which the diameter was increased to some 45 mm The interior of the ascending aorta showed extensive yellow mottling in patches, some separate and some discrete, 3-4 mm in diameter Near the root the mottling formed an arborescent pattern The surface of these mottled areas was slightly raised In thickness the wall measured 3 5-4 mm, but in places it measured 6 mm On the posterior wall of the interior of the aorta was a large yellowish raised patch beginning about 35 mm from the root of the aorta and extending beyond the level at which the vessel had been cut In this area there was very considerable thickening of subintimal tissue and the tunica intima readily separated off from the other coats The root of the aorta showed comparatively little pathological change There were small thin yellow patches of atheroma round the root of the right coronary artery and adjacent to the orifice of the left coronary artery, but the lumen of these vessels was not materially narrowed Except for the large area mentioned, the wall of the aorta was pliable and showed no general pathological alteration, there being only slight subintimal atheroma

Section of the patches on the wall showed atheromatous changes of the intimal and subintimal coats External to the smooth endothelium of the intima was a firm, pale yellow layer some 3 mm in thickness, external to this the darker coloured and almost unchanged tunica media There was little, if any, calcification in the subintimal thickenings

. . . .

Coronary arteries.—Both of the arteries and their branches were the seat of advanced and widespread degenerative changes, which had caused thickening of the wall of, especially, the medium-sized and smaller vessels, and diminution of their lumen. The arteries most affected were those in the anterior ventricular furrow, of which there were two, one from each coronary stem. These vessels were so thickened and calcified that their lumen was almost obliterated. A recently occluded vessel was not found as a cause for the infarction mentioned above.

Right coronary artery.—Near its root the external diameter of this vessel was 9 mm. Its wall was greatly thickened and the lumen, oval in outline, measured 2 by 3.6 mm. The thickening involved mainly the subintimal coat and also the tunica media, and the wall was firm and rigid. Section of the wall showed patches of degenerated cheesy material in the centre of the thickened areas, and the changes involved almost the whole circumference of the vessel.

From near the root of the artery a branch, 4 mm. in diameter, passed in the anterior interventricular sulcus to the inferior margin, lying by the side of a slightly larger branch from the stem of the left coronary artery. The two vessels ran side by side in the anterior longitudinal furrow, the left one giving a superficial branch and then entering the muscular coat half-way down, while the right artery ran onwards superficially. The wall of both of these vessels was greatly thickened, and the lumen in each reduced to a minute capillary cleft.

Half an inch from its root the diameter of the right coronary artery was 8 mm. At this point the thickening was less pronounced and the lumen wider. The artery continued as a large vessel and gave off numerous branches, a small branch in the epicardial fat along the right margin, a very tortuous branch which ran on the inferior surface an inch from the right margin, a small vessel to the base of the ventricle, at the left portion of the coronary sulcus three branches to the inferior surface of the ventricle, arising close to one another and measuring 2-3 mm. in diameter, finally, the terminal portion of the artery turned downwards in the inferior interventricular sulcus. The distal portion of the artery showed much slighter pathological change, the lumen, though diminished, being distinct.

The left coronary artery was smaller than the right, and its wall was less affected by pathological change. The external diameter at the root was 6 mm. and the wall not more than 1 mm. thick. It gave off a large branch already mentioned to the anterior interventricular furrow, the wall of this branch was more affected pathologically than the stem of the artery, there being marked intimal thickening in patches near the root, while an inch or two distally the thickening was even more pronounced, involving the whole wall and reducing the lumen to an extremely small

size The left coronary artery gave a large branch to the left margin of the heart The wall of this branch too was distinctly thickened, and in the more distal portion so thickened as to reduce the lumen to the smallest dimensions

THE CONDITION OF THE HEART IN RELATION TO THE SYMPTOMS

(1) The first signs of heart impairment occurred in 1908, seventeen years before his death, when he experienced a sharp attack of severe pain, which with our present knowledge would be diagnosed as due to a coronary thrombosis There is evidence in the heart to confirm this view, for the patch of fibrosis near the apex corresponds to the structural damage which would be caused by such an attack

Sir Thomas Lewis, who was good enough to send me his opinion after examining the heart, wrote to me as follows

"Grant and I examined the heart very closely and we are agreed that there are amply sufficient old-standing changes at the apex of the heart to account for the first attack of pain described in his case notes That attack of pain is strongly suggestive of coronary thrombosis, and the fibrosis at the apex is distributed in a way that also suggests thrombotic obstruction of an apical branch "

(2) The severe atheroma of the coronary arteries and their branches, with diminution of the lumen affords ample cause for the occurrence of attacks of cardiac pain Both of the arteries were affected and the anterior interventricular branch of each was greatly narrowed

(3) There were numerous small patches of cicatrization in the substance of the muscular wall of the left ventricle These patches though smaller were of the same nature as the larger fibrous patch at the apex, which was due to thrombosis of an apical branch Other arteries to the left ventricle were profoundly altered and their lumen narrowed This has been shown for example in the marginal artery of the left ventricle There would therefore appear to have been numerous small thromboses at different times, each of which would be accompanied by symptoms similar to those experienced at the first attack Several such attacks are recorded and the similarity is brought out in the case history It is noted, for example, that on many occasions the attacks came on during rest and were quite unrelated to effort

(4) The presence of numerous small blood vessels on the surface of the heart points to there having been an opening up of small vessels and the establishment of at least a partial anastomotic pathway for the supply of blood to the areas most severely impaired by the attacks of thrombosis In this connection it may be remarked that during the last few months

of his life between August 1924 and January 1925, Sir James's condition showed slight improvement I found, for example, that not only could he walk for some distance down Exhibition Road from his home in Albert Hall Mansions, but he was able to walk up that road without distress, though at a slow pace This improvement doubtless was due to a slight improvement, by anastomosis, in the arterial supply to the heart

(5) The terminal severe attack of pain and cardiac impairment was associated with the occurrence of the recent infarction found at the apex of the left ventricle

(6) There was no evidence of any impairment in the valves or in the genetic system of the heart The impairment was entirely in the muscular wall, brought about by the atheromatous disease of the arteries

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1909

SIR WILLIAM OSLER

DESCRIPTION OF THE SKIN NODULES IN SUBACUTE BACTERIAL ENDOCARDITIS, LATER TO BE KNOWN AS OSLER NODES

SIR WILLIAM OSLER

After a crayon portrait by the American artist, John Singer Sargent Reproduced by permission of the College of Physicians of Philadelphia

SIR WILLIAM OSLER

(1849-1919)

"I have loved no darkness
Sophisticated no truth
Nursed no delusion
Allowed no fear."

—William Osler

WILLIAM OSLER was the son of a clergyman, the Reverend Featherstone Lake Osler, and of Ellen Free Pickton Osler. He was born, the sixth son in a family of nine children, in the parsonage at Bond Head, Ontario, near the lower edge of the upper Canadian wilderness, on July 12, 1849.

Young Osler gained his early education at the local grammar school in Dundas, a town of 3,000 people, to which his family had moved in 1857. For some boyish prank Osler was expelled from this school in June of 1864. The next autumn, following in the footsteps of his brothers, he was sent to a boarding school at Barrie. At Barrie, Osler excelled in sports and at one time won the school prize for kicking the football the longest distance. And at Barrie, accompanied by a friend, he swam one and a half miles across Kempenfelt Bay, a feat rendered difficult by the chilliness of the water.

Because Barrie was far from his home in Dundas, Osler, in 1866, was sent to study at the Episcopal school at Weston. Under the influence of the Reverend William A. Johnson, this school had become known as the Trinity College School, and was preparatory school for Trinity University. Osler derived much benefit from his association with Johnson, whom he greatly admired; among other things, he was introduced by Johnson to microscopy. There, also, young Osler became acquainted with Dr. James Bovell, the medical director of Trinity College.

Osler matriculated at Trinity University in the fall of 1867, following his winning of one of the Dixon Prize Scholarships at Weston. During the spring quarter of 1868, Osler indicated his interest in medicine by attending the medical school in the afternoons. In the fall of 1868 he abandoned his course in liberal arts and studied medicine at the Toronto Medical School.

While he was a medical student, Osler published his first paper, "Christmas and the Microscope." It appeared in Hardwicke's "Science-Gossip" for February, 1869. In that paper the young student of microscopy enumerated the living things in a bottle of spring water which he was able to identify with the aid of a microscope. Such was Osler's modest beginning of an extraordinary career in letters.

In 1870 Osler decided to study medicine in Montreal at the McGill Medical School. There he worked under four brilliant men: R. Palmer Howard, Adam H. Wright, Duncan MacCallum, and J. Morley Drake. Three of his teachers had studied under Robert Graves and William Stokes at Edinburgh, and indeed, McGill closely followed the educational methods of the famous Scotch school. Osler was graduated from McGill in 1872, winning a special prize for his thesis.

Osler spent the next two years in postgraduate study in Great Britain. For several months he worked in the laboratory of John Burdon Sanderson at the University College Hospital of London. Thirty-four years later he was destined to succeed Sander-

son as Regius professor of medicine at Oxford The results of some of his studies in Sanderson's laboratory on hematologic problems were read before the Royal Microscopical Society and subsequently were published in its journal [1]

For the next few months Osler continued his hematologic studies and under the microscope observed the peculiar globoid bodies now known as blood platelets Previous investigators had observed these bodies but none of his predecessors actually had seen them in the circulating blood To Osler belongs the credit of establishing this important contribution to medicine on a firm basis

Leaving England Osler traveled on the Continent and studied at the medical centers in Berlin and Vienna He returned to London for a few months and then embarked for his native country Soon after returning to Canada, Osler accepted an appointment at McGill University as lecturer on the institutes of medicine (1874) In 1876 he was appointed to the newly created post of pathologist at the Montreal General Hospital, and in the spring of 1878 he succeeded Drake as full-time physician in the same hospital

Osler returned to London for a short visit in 1878 and qualified for and received his membership in the Royal College of Physicians The same year he attended the meeting of the British Medical Association and there, as Cushing wrote, no doubt became acquainted with Sir T Grainger Stewart, Sir Jonathan Hutchinson, Sir Clifford Allbutt, Sir William Gairdner, and Sir William Broadbent, who later were to become his staunch friends

Early in the autumn of 1878, Osler returned to McGill University to resume his medical teaching He had felt for some time that one of the errors in the practice of medicine was overemphasis on the use of drugs in the treatment of disease For this reason he believed with Hahnemann that the natural tendency of disease was toward recovery, provided that the patient was decently cared for, properly nursed, and not overdosed Therefore, when Osler returned as full-time physician to McGill General Hospital he adopted therapeutic methods which were a revelation to the older physicans in the hospital

In the spring of 1880, Osler, who had been greatly interested in the principles of the physiology of digestion made possible by Beaumont's researches on Alexis St Martin, desired to perform a necropsy on his body when he learned of St Martin's demise It was Osler's intention to preserve St Martin's stomach and send it to the United States Army Medical Museum in Washington, D C The relatives of St Martin refused Osler the permission for a necropsy The refusal was a great disappointment to him

In 1881, Osler and R Palmer Howard represented McGill University at the meeting of the International Medical Congress held in London under the presidency of Sir James Paget It was the privilege of those in attendance to hear excellent addresses by Paget, Virchow, John Shaw Billings, Huxley, and Pasteur Osler profited much from these meetings and probably his contact there with Billings encouraged Osler in several important bibliographic undertakings and in his later interest in medical libraries Osler read a paper at this congress

Following another trip to London in 1884, Osler accepted the chair of clinical medicine at the University of Pennsylvania, and he remained in the United States until 1905 In Philadelphia, he made many friends including S W Gross, Minis Hays, James Wilson, and Weir Mitchell

Through much clinical and pathologic study at Montreal, Osler had come to believe that endocarditis was bacterial in origin In Philadelphia he continued his re-

[1]Osler, William Action of certain reagents—atropia physostigma and curare—on the colourless blood-corpuscles Quart J Microscopical Sc 13 307-309 1873

search in this subject and the studies he made there formed the basis for his Goulstonian lectures, which he delivered in London in 1885 In August of that same year Osler, as president of the Canadian Medical Association, gave the annual address at the meeting of the association in Chatham, Western Ontario

Osler soon was elected to membership in the College of Physicians of Philadelphia and there he made much use of that organization's library, which at that time contained more than 34,000 volumes He served for five years as a member of the library committee of the College and under his auspices many precious books were added to the library

In 1889, Osler accepted the offer from the newly opened Johns Hopkins Hospital in Baltimore to serve as physician-in-chief He assumed his new duties in May, and on him fell the responsibility of organizing the new clinic Other chiefs-of-staff who were also destined to make Johns Hopkins a famous medical center were William Welch, W S Halstead, Henry M Hurd, and Howard A Kelly

In 1891 Osler began to write his great work, "The Principles and Practice of Medicine," first published in 1892 The book filled a decided need and was eagerly received by the medical profession in the United States, Canada, and England Edition after edition of this popular and important work was published and later, under the editorship of McCrae, it continued in many editions It is still being published under the editorship of Henry A Christian and still is the standard textbook in many medical schools

One of the things that Osler missed in Baltimore was the fine library of the College of Physicians of Philadelphia In 1891 he volunteered to serve on the library committee of the then almost defunct Medical and Chirurgical Faculty of Maryland, which had a library of a few hundred musty tomes He aided in the rejuvenation of the faculty and kept his membership on the library committee until his departure for England in 1905, and saw the library grow to the size of 15,000 volumes

Osler kept his interest in books and libraries throughout his life and contributed financial and moral support to the libraries of McGill, the United States Surgeon General's Office, the College of Physicians of Philadelphia, and to his old preparatory school of Weston, Trinity College School To many others he made important gifts

On May 7, 1892, Osler married Grace Revere Gross, the widow of his former friend, Dr Samuel W Gross, who had died in April, 1889 Dr and Mrs Osler spent the summer of 1892 in England, but even on his honeymoon he could not be kept away from medical meetings

They returned to Baltimore in August, 1892 On October 4 of that same year, Osler gave an address in Minneapolis on the occasion of the opening of the new medical buildings at the University of Minnesota The following day he addressed the Minnesota Academy of Medicine choosing as his subject, the "License to Practice "[2]

In December, 1892, Osler received a splendid offer from McGill University, but did not accept it and devoted himself to problems of organization connected with the opening of the new medical school at Johns Hopkins University in Baltimore

The ensuing years at Johns Hopkins were extremely busy ones for the chief of the medical staff He was called on countless times to deliver speeches, to write many medical and literary contributions, and to lecture to undergraduate and postgraduate students

Among the many honors that he won, Osler especially cherished his election, in 1898, to the Royal Society Election came as a complete surprise to him It happened that at the time Lord Lister was president of the world-famous body In the same year Osler became dean of the School of Medicine of Johns Hopkins University

[2]Northwest Lancet 12 383, 1892

In the summer of 1898, Osler made another trip to Great Britain During his stay he was awarded two honorary degrees of Doctor of Laws, one from the University of Aberdeen and one from the University of Edinburgh

Osler had, for many years, wished to make his home in Great Britain, and had been tempted by the desires of many of his British friends to be a candidate for the chair of medicine at the University of Edinburgh The chair became vacant in 1900, at which time Osler did become a candidate, but he reconsidered and withdrew his application In August, 1904, however, being offered the position of Regius professor of medicine at the University of Oxford, he accepted

Osler assumed his new office in the latter part of May, 1905 His first official duty was to act as a curator, ex officio, of the Bodleian Library, a task, we may assume, from which he derived much pleasure In a special convocation in June, 1905, he received from Oxford University the degree, Doctor of Science

On October 18, 1906, Osler gave the annual Harveian oration He chose for his subject, ''The Growth of Truth as Illustrated in the Discovery of the Circulation of the Blood ''

In 1907 appeared the first three volumes of Osler's ''System of Modern Medicine,'' which was to become an epoch-making contribution to medicine

In 1908 he was chosen to give the Linacre lecture This he delivered on May 6 at St John's College, Cambridge, choosing the life of Thomas Linacre as his subject On June 12 he read before the Association of Physicians of Great Britain and Ireland his classic account of ''Chronic Infectious Endocarditis '' This contains the description of the ''Osler nodes '' We are presenting this account to our readers

In the spring of 1910, Osler delivered the famous Lumleian lectures before the Royal College of Physicians He chose angina pectoris as the subject of his address In 1911 at the Coronation of King George, Osler was created a baronet

Osler made his last visit to the United States in 1913 To students of medical history this was an auspicious occasion, for in that year he gave a course of lectures on ''The Evolution of Modern Medicine,'' the Silliman Lectures, at Yale University These were later published in book form and constitute one of his best works That year also, he served as president of the medical section of the International Medical Congress at its meeting in London

In 1914 Osler was elected president of the Bibliographical Society He chose as the subject of his address ''The Earliest Printed Medical Books '' This was published as a preface, nearly four years after his death, to his important bibliographic study, ''Incunabula Medica'' (1923)

During the World War, Osler served as a civilian member of the committees at the War Office He also served on the Committee for the Medical History of the War and on the War Reports Committee He held the position of honorary colonel in the army and helped greatly in building up the morale of the British, Canadian, and American soldiers who returned wounded from the front

It had been Osler's philosophy to accept the sorrows and the joys of the world with equanimity, but the death of his only son, Revere, who had been severely wounded in France, occurred in August, 1917, and was a blow very hard for him to sustain

In July, 1919, Osler celebrated his seventieth birthday He had suffered many attacks of bronchopneumonia, which left him very weak The exertions of his last few years, the heart-breaking loss of his son, and the heavy strain of the war all contributed to the undermining of his health His final illness was protracted influenza, to which he succumbed on December 29, 1919

CHRONIC INFECTIOUS ENDOCARDITIS*

By

WILLIAM OSLER

[Description of Osler Nodes]

AN ENDOCARDITIS with fever as its only symptom may be prolonged for weeks or months under many different circumstances. Following rheumatic fever in a child an endocardial complication may keep up a temperature of from 100° to 101° for several months, during which time there may be no other symptoms and the general condition may remain fairly good. In chronic valvular disease in the stage of broken compensation slight irregular fever may persist for months, associated with the presence of fresh endocarditis. As a rule, the form of endocarditis to which we give the term infective, septic, or ulcerative runs its course under three months. That occasional instances were characterized by a very protracted course was noted by Wilks, Bristowe, Coupland, and Lancereaux. In my Goulstonian Lectures, 1885, I stated that this type had the following characteristics. The fever was irregular and intermittent, resembling ague; the cold, hot, and sweating stages might succeed each other with great regularity; in the intervals fever might be absent; two or three paroxysms could occur in the course of a day. In many of the instances the disease was prolonged to three or four months, and I give the notes of a case of Bristowe's, in which the condition persisted for five months. The recurring chills usually led to the diagnosis of malaria and also gave rise to the opinion widely held, particularly by French writers, that ulcerative endocarditis could be caused by this disease. The cases to which I wish to call attention in this communication are of this chronic character, not marked specially by chills, but by a protracted fever, often not very high but from four to twelve months' duration. At the time of the delivery of the Goulstonian Lectures I had not seen a case of this type. In the past twenty years I have seen ten cases of this form, two of which I have already reported (*Practitioner*, 1893).

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It has long been recognized that malignant endocarditis is really an acute septicaemia with localization on the endocardium, but the symptoms are not necessarily due to the local lesion. The clinical picture is a

*Read at the Association of Physicians of Great Britain and Ireland, Edinburgh, June 12, 1908. Printed in Quart. J. Med. 2: 219-230, 1909.

septicaemia sometimes of a typhoid type, sometimes like a pyaemia—then again with predominant meningeal symptoms, occasionally with pronounced cardiac features. The pneumococcic, the gonorrheal, and the streptococcic forms present, as a rule, a picture in which the heart-symptoms are in the background. Cases of infection with these organisms may run an identical course without any endocarditis. On the other hand, there is a large group of cases in which the endocarditis plays a more important role and the vegetations and ulcerations appear to be directly responsible for the fever and the associated symptoms. As a rule, the valves involved are already the seat of a sclerotic change. The source of the infection is rarely to be determined. Thus, in only one of the series here reported was there an external lesion. The patients in this series were all adults, five women and five men. In six there was a past history of rheumatic fever, eight had old mitral lesions, two aortic, well compensated, and not giving any trouble at the time of the onset of the symptoms. It was not always possible to get a definite history of how the attacks began. In five of the cases there were chills and fever, mistaken for malaria. Cough and loss of weight in some cases suggested tuberculosis. The slight fever without any localizing symptoms may raise the suspicion of typhoid fever. In my series these have been the three diseases the diagnosis of which has been suggested. Once established the fever becomes the dominant, and for months may be the only, symptom. This is the most striking peculiarity of the cases. Week after week, month after month, the daily rise of one and a half or two degrees may be the only indication there is of an existing mischief. In Case I, in which the fever lasted for thirteen months, the patient's sister, a trained nurse, had decorated the room with yards of the temperature charts, fever with an occasional sweat were the only symptoms. The appetite remained good and she lost very little in weight. There were no embolic features and from month to month there were few, if any, changes in the cardiac condition. In this very protracted form chills are not nearly so common as in the more acute cases, nor is the fever so high, not often reaching above 102.5° or 103°. It is of a remittent type, not falling to normal at any period of the day. With the occurrence of a chill the temperature may rise to 104° or 105°, but in none of the cases was there the type of fever in which the paroxysms recur with great regularity—quotidian or tertian, as we see so often in the acute forms of ulcerative endocarditis. Another peculiarity is the occurrence of periods of apyrexia, usually towards the end, but in one or two of the cases there were afebrile interludes which gave deceptive promise of recovery. It is well recognized now that fever is not an invariable accompaniment of endocarditis. Following pneumonia there may be for months a slight toxaemia with little or no fever in connexion with a patch of endocarditis.

The cardiac features in this group are usually well marked, but as a rule there are no symptoms. The patients complain neither of palpitation

nor of pain There is no dyspnoea except towards the close, and in no case did dropsy occur In eight of the ten cases there were the well-marked physical signs of a mitral lesion and the associated slight enlargement of the heart In only six cases was there marked hypertrophy and dilatation In two of the cases there was aortic insufficiency One of the most striking circumstances is the very slight change in the character of the heart murmur in spite of the fact of most extensive vegetations and alterations in the valves Thus in the case of Dr R T, with the condition of whose heart I had been familiar for fourteen years, the comparison between my first examination in 1889 and that in 1893 showed very little change beyond the slightly greater dislocation outwards of the apex beat In several of the cases the absence of any change in the character of the heart murmur and the remarkably quiet, negative state of the organ were urged strongly against the existence of endocarditis It is rather remarkable, considering the anatomical changes, that so little alteration may occur in the physical signs In Case VI, Dr B T, the murmur of aortic insufficiency became more intense towards the close, but in no instance was there the development under observation of alterations in the physical signs such as are sometimes seen in acute ulcerative endocarditis

Embolism, to cause symptoms, occurred in four cases of the series—in Cases III, IV, and IX in the brain with haemiplegia, Case VIII in the retinal arteries and in the spleen and kidneys This is in striking contrast to the frequency of this complication in the more acute types of endocarditis

One of the most interesting features of the disease and one to which very little attention has been paid is the occurrence of ephemeral spots of a painful nodular erythema, chiefly in the skin of the hands and feet, the *nodosités cutanées éphémères* of the French My attention was first called to these in the patient of Dr Mullen of Hamilton, whose description is admirable "The spots came out at intervals as small swollen areas, some the size of a pea, others a centimetre and a half in diameter, raised, red, with a whitish point in the centre I have known them to pass away in a few hours, but more commonly they last for a day, or even longer The commonest situation is near the tip of the finger, which may be slightly swollen" Spots of this character occurred in seven of the cases and in three at least they were of importance in determining the diagnosis Thus in the case of Dr Carroll, the well-known American Army Surgeon, the collaborator with Dr Reed in the brilliant work upon yellow fever, the presence of these spots appeared to me to clinch the diagnosis They are not beneath but in the skin and they are not unlike an ordinary wheal of urticaria The pads of the fingers and toes, the thenar and hyperthenar eminences, the sides of the fingers, and the skin of the lower part of the arm are the most common localities In one case they were present in the skin of the flank I have never seen them haemorrhagic, but always erythematous, sometimes of a very vivid pink hue, with a slightly opaque centre

The diagnosis in this group of cases may offer great difficulties For weeks, indeed for several months, there may be only fever, and unless there have been special features pointing to the heart, such as the development of a diastolic murmur or the great intensification of a mitral bruit, it may be impossible to settle the diagnosis There are, indeed, cases in which from beginning to close no heart murmur has been present By far the most suggestive features are (1) a knowledge of the existence of an old valve lesion This was present in every one of my series (2) The occurrence of embolic features, sudden swelling of the spleen, with friction in the left flank, sudden attack of haematuria, embolism of the retinal arteries, hemiplegia or the blocking of a vessel in one of the limbs (3) The onset of special skin symptoms, purpura, and more particularly the painful erythematous nodules to which I have referred Present in seven of the ten cases, these are of definite diagnostic import They are in all probability caused by minute emboli (4) The progressive cardiac changes, the gradual increase in the dilatation of the heart, the marked change in the character of a mitral murmur, the onset of a loud rasping tricuspid murmur, or the development under observation of an aortic diastolic bruit

With carefully made blood-cultures one should now be able to determine the presence of the septicaemia This was easily done in three of my more recent cases An onset with chills and fever and slight swelling of the spleen almost always leads to the diagnosis of malaria, more particularly in regions in which this disease prevails, but in not one of my cases was there any difficulty in excluding this by careful microscopical examination of the blood It was not always possible to convince the physician With slight cough tuberculosis may be suspected, as happened in two or three cases of my series For many weeks the patient may present nothing but a pyrexia, of doubtful origin, or a cryptogenetic septicaemia and as he may look very well and may feel very well, and there are no special symptoms, and with a heart-condition that may have remained unchanged for years, it is not easy to reach a positive diagnosis The blood-cultures and the presence of the painful erythematous nodules and the occurrence of embolism furnish the most important aids

The anatomical condition in these cases is quite unlike that of the ordinary ulcerative endocarditis In the three specimens I have had an opportunity of studying there was no actual ulceration, but large proliferative vegetations, firm and hard, greyish yellow in colour, projected from the endocardium of the valves like large condylomata, encrusting the chordae tendineae and extending to the endocardium of the auricle The condition is quite unlike the globose vegetations of the pneumococcal and gonorrhoeal endocarditis or the superficial ulcerative erosions of the acute septic cases

The organisms responsible for this condition have been carefully studied In my series cultures were made in six cases In three they were negative In two streptococci were present, in one a staphylococcus

While, as a rule, this condition is much more commonly caused by the streptococcus other organisms may be present Thus Fraenkel has reported one instance of a pneumococcus endocarditis persisting for nearly six months (*Deutsche med Woch* , 1900) Of sixteen cases of this chronic form, the clinical course of which extended from four to eight months, Harbitz (*Deutsche med Woch*, 1899) found pneumococci in four, streptococci in nine, and in eight other micro-organisms Lenhartz (*Deutsche med Woch*, 1901), who has reported sixteen cases with a duration of from three to seven months, found staphylococci and streptococci the common organisms, the pneumococcus once and the gonococcus once In the majority of cases it seems to be a mild streptococcus infection, possibly by a special form Possibly in some instances there may be a special resistance on the part of the host, but these are points which must be settled by future investigations These are cases in which the possibility of successful vaccine treatment should be considered It was tried in two cases of my series, but in both rather late, and in neither did it seem to have special influence Horder has treated a case of this chronic type with a vaccine prepared from the patient's organism, but without success The results in the acute forms are discussed by him in the *Practitioner*, May, 1908

1912

JAMES B HERRICK

DESCRIPTION OF CORONARY THROMBOSIS

JAMES BRYAN HERRICK

(Courtesy Central Interurban Clinical Club)

JAMES BRYAN HERRICK

(1861- —)

"Men succeed because of native ability and in small measure because of chance, but chiefly through hard work, through a knowledge of their special vocational subject and through their ability to apply this knowledge" —J B Herrick, in his essay, "N S Davis"

JAMES BRYAN HERRICK, a living member of the great company of classic cardiologists, whose outstanding contribution to this field we are reprinting herein, was born on August 11, 1861, in Oak Park, Illinois, the son of Origen White Herrick and Dora E Herrick His maternal grandfather operated a saw mill on the Desplaines River, and his mother, the former Dora E Kettlestrings, who was also born in Oak Park was (in 1935) the oldest native daughter of that village Her father immigrated to the United States from England in 1833, traveled westward in a covered wagon and settled on a homestead on the site which is now Oak Park

James Herrick attended the Oak Park High School and the Rock River Seminary at Mount Morris, Illinois He received his collegiate training at the University of Michigan, where he was graduated with the degree of Bachelor of Arts in 1882 At Michigan, young Herrick came under the influence of Moses Coit Tyler, professor of English literature Tyler was not only an authority on American literature, having published the best history of American literature of the colonial and revolutionary periods, but also was a Chaucerian scholar of the highest order Under Tyler's influence, Herrick was instilled with a profound appreciation of Chaucer that has endured [1]

Herrick began the study of medicine in 1886, choosing Rush Medical College as his school He was graduated from Rush in 1888 with the degree of Doctor of Medicine, and he interned at the Cook County Hospital In 1889 he married Zellah P Davis, of his native town of Oak Park

In addition to making important contributions to medical literature at an early date, Herrick found time to pursue literary activities He also acted as an officer of the board of the Lewis Institute and the McCormick Memorial Institute At this writing, he is secretary of the board of trustees of the Lewis Institute and is president of the McCormick Memorial Institute in Chicago

One year (1889) after his graduation from Rush, Herrick contributed three articles to medical literature The first of these was "A Case of Hemophilia Neonatorum" This appeared in the first volume of the "North American Practitioner" By a typographical error, the article was credited to James B Henrick. In the same volume Herrick reported on traumatic rupture of the bladder In the "Western Medical Reporter" he described an operation for sacro-iliac tuberculosis From 1889 until 1935, according to Holmes, there have been only five individual years in which Herrick did not contribute to scientific literature In 1896 he published eleven articles, including three on cardiovascular disease and two on anemia

In 1904 Herrick made an original observation in hematology At that time a negro who had a sore on his ankle and evidences of previous scarring presented himself for treatment In making a hematologic examination Herrick discovered that the blood of this patient showed numerous elongated or sickle-shaped red cells Although the discovery was made in 1904 and was confirmed in 1906 by E E Irons, Herrick waited until 1910 to publish it [2] In 1910 also appeared his first article on angina pectoris—

[1] Herrick's appreciation is well expressed in his recent article, Why I Read Chaucer at 70 Ann Med Hist 5 62-72 1933

[2] Herrick J B Peculiar elongated and sickle-shape red blood corpuscles in a case of severe anemia Arch Int Med 6 517-521, 1910

a disease on which he later made many scholarly contributions, and with which his name probably will be associated in the future

Herrick worked hard to make the profession recognize the importance of coronary thrombosis, and in 1912, the "Journal of the American Medical Association" published his classical account of this condition in a paper entitled "Clinical Features of Sudden Obstruction of the Coronary Arteries" Besides giving to the medical world by far the best extant description of this disease, Herrick showed that sudden obstruction of a coronary artery is not necessarily fatal We consider it a special privilege to reprint in full this historic classic It is of significance to note, in this connection, that in January, 1939, Herrick[3] pointed out that Robert Adams about 100 years ago came very close to the discovery of coronary thrombosis Adam Hammer described the first case of coronary thrombosis with correct diagnosis ante mortem in 1878,[4] and the first completed description of the disease was published in 1910 by Obrastzow and Straschesko[5]

In 1918 Herrick and Nuzum[6] made the first direct reference to the occurrence of anginal pain among patients having severe anemia

Since 1918 Herrick has made many contributions to the literature of medicine The reader is referred to the bibliography of 135 articles by Herrick compiled by Elizabeth Carr, librarian of the Northwestern University Medical School This constitutes an appendix to William H Holmes' appreciative study of Herrick, from which study the data in the present brief biographic account were, in part, obtained

Dr Herrick has received many honors He is past president of the Chicago Pathological Society, he was the first (1915) president of the Chicago Society of Internal Medicine, he has served as president (1925) of the Institute of Medicine of Chicago and of the Association of American Physicians (1923), and he was president of the American Heart Association in 1927 In 1938 he was again honored by the Association of American Physicians by being elected vice-president of that distinguished group of internists His services with the McCormick Memorial Institute and the Lewis Institute have already been mentioned As this is written (1940), he is president of the Congress of American Physicians and Surgeons

The University of Michigan has twice honored her distinguished son in 1907, with an honorary degree of Master of Arts, and in 1932, with the degree of Doctor of Laws

Dr Herrick has been affiliated with many institutions in many professional capacities He was instructor in medicine at Rush Medical College from 1890 to 1894, assistant professor of medicine at the same school from 1894 to 1900, and professor of medicine from 1900 to 1926 Since 1926 he has been emeritus professor in the same college He also was professor of the theory and practice of medicine and professor of materia medica and therapy at the old Northwestern University Women's Medical School, which became extinct in 1902 Since 1898 he has been attending physician to the Presbyterian Hospital in Chicago He is an honorary fellow of the New York Academy of Medicine, and in 1931 was selected to be the lecturer of the Harvey Society of that institution In 1930 he was awarded the George M Kober Medal conferred by the Association of American Physicians In 1939, at the ninetieth annual meeting of the American Medical Association, Dr Herrick received the second Distinguished Service Medal to be awarded by that association, the first having been awarded in 1938 to Dr Rudolph Matas of New Orleans

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[3]Herrick J B Robert Adams *Surgeon* and his contributions to cardiology Ann Med Hist. 1 45-49 (Jan) 1939

[4]Hammer Adam Ein Fall von thrombotischem Verschlusse einer der Kranzarterien des Herzens Am Krankenbette konstatirt, Wien med Wchnschr 28 97-102 1878

[5]Obrastzow W P and Straschesko N D Zur Kenntnis der Thrombose der Koronararterien des Herzens Ztschr f klin Med 71 116-132 1910

[6]Herrick J B and Nuzum F R Angina pectoris *J A M A 70 67-70 1918*

CLINICAL FEATURES OF SUDDEN OBSTRUCTION OF THE CORONARY ARTERIES*

By

JAMES B. HERRICK, M. D.

Chicago

OBSTRUCTION of a coronary artery or any of its large branches has long been regarded as a serious accident. Several events contributed toward the prevalence of the view that this condition was almost always suddenly fatal. Parry's writings on angina pectoris and its relation to coronary disease, Jenner's observations on the same condition centering about John Hunter's case, Thorvaldsen's tragic death in the theater in Copenhagen with the finding of a plugged coronary, sharply attracted attention to the relation between the coronary and sudden death. In Germany Cohnheim supported the views of Hyrtl and Henle as to lack of considerable anastomosis, and as late as 1881 lent the influence of his name to the doctrine that the coronary arteries were end-arteries, his Leipsic necropsy experience, as well as experiments on dogs, forced him to conclude that the sudden occlusion of one of these vessels or of one of the larger branches, such as the ramus descendens of the left coronary, meant death within a few minutes. Others emphasized the same view.

No one at all familiar with the clinical, pathologic or experimental features of cardiac disease can question the importance of the coronaries. The influence of sclerosis of these vessels in the way of producing anemic necrosis and fibrosis of the myocardium, with such possible results as aneurysm, rupture or dilatation of the heart, is well known. So also is the relation of the coronaries to many cases of angina pectoris, and to cardiac disturbances rather indefinitely classed as chronic myocarditis, cardiac irregularities, etc. It must be admitted, also, that the reputation of the descending branch of the left coronary as the artery of sudden death is not undeserved.

But there are reasons for believing that even large branches of the coronary arteries may be occluded—at times acutely occluded—without resulting death, at least without death in the immediate future. Even the main trunk may at times be obstructed and the patient live. It is the object of this paper to present a few facts along this line, and particularly to describe some of the clinical manifestations of sudden yet not immediately fatal cases of coronary obstruction.

*J. A. M. A. 59: 2015-2020, 1912.

Before presenting the clinical features of coronary obstruction, it may be well to consider certain facts that go to prove that sudden obstruction is not necessarily fatal Such proof is afforded by a study of the anatomy of the normal as well as of the diseased heart, by animal experiment and by bedside experience

The coronaries are not so strictly end-arteries, i e, with merely capillary anastomoses, as Cohnheim and others thought By careful dissections, by injection of one artery from another, by skiagraphs of injected arteries and by direct inspection of hearts made translucent by special methods, there is proof of an anatomic anastomosis that is by no means negligible

Jamin and Merkel's* beautiful stereoscopic skiagrams show the remarkably rich blood-supply of the heart, with occasional anastomoses between vessels of considerable size The possibility of injection of the coronary artery from the other is admitted even by those who deny that such injection proves more than a capillary non-functioning anastomosis Amenomiya,[1] by injecting hearts of young persons, showed naked-eye anastomoses in the subepicardial tissue He feels that Hirsch and Spalteholz[2] have nearly cleared up the question as to the relation between the heart muscle and disease of the coronary artery from the anatomic standpoint Hirsch says that in dogs the anastomosing vessels are functionally competent, and Spalteholz says that in man the vessels are nearly the same as in dogs, rich in anastomoses even in those of considerable caliber The latter investigator, by a method of injection and treatment of the heart so as to make the muscle transparent, shows to the naked eye that there are anastomoses of considerable size

Among others who are on record as believing that there are non-negligible anastomoses may be mentioned Haller, Huchard, Orth, Michaelis, Langer, Legg, West All recognize, however, that there are individual differences and also that though the heart may show rich anastomoses, these are not necessarily functional, i e, that an artery which anatomically is not a terminal artery may yet be such functionally

But there is proof not only of anatomic connection between the two coronaries, but that in certain instances, at least, such connection is of functional value Experiments on lower animals and the clinical experiment of disease of the coronaries with autopsy findings show this

Much of the earlier experimental work on the lower animals, obstructing the coronary arteries by ligatures, clamps or artificial emboli, gave promptly fatal result Among those who worked along this line and

*Jamin and Merkel Die Koronararterien des menschlichen Herzens in stereoskopischen Rontgenbildern Jena 1907 Extensive bibliographies are contained in the articles by Thorel (Lubarsch-Ostertag s Ergebnisse ix Abt 1) and in Amenomiya (Virchows Arch f path Anat 1910 cxcix 187) I repeat only some of the more important references and add new ones

[1] Amenomiya Ueber die Beziehungen zwischen Koronararterien und Papillarmuskeln im Herzen Virchows Arch f path Anat 1910 cxcix 187

[2] Hirsch and Spalteholz Koronararterien und Herzmuskel Deutsch med Wchnschr 1907 No 20

reached these conclusions may be mentioned Erichsen (1842), Panum (1862), von Bezold, Samuelson (1880), Cohnheim and Schulthess-Rechberg (1881), G. Sée, Rochefontaine and Roussy (1881), Bettelheim (1892), Kronecker, and, to some extent, Michaelis. The work of Cohnheim[3] attracted particular attention and his conclusions as to end-arteries, irreparable injury, and cessation of the beat of both sides of the heart within two minutes from the time of shutting off the coronary circulation confirmed and elaborated the conclusions of the earlier experimenters, and was in turn confirmed by the French writers just named, by Bettelheim and others.

But soon dissent was heard from various quarters as to many of Cohnheim's results, and among other things as to the sudden death following the ligations. Michaelis found that the injury from ligation in rabbits was not so serious or irreparable as in dogs. Fenoglio and Drouguell, in 1888, found that some dogs might live. Porter showed that after ligation of one or two large branches of the coronary artery a dog might live hours or days. More than half his animals lived after ligation of the descending branch of the left coronary. Von Frey, at the Congress for Internal Medicine in 1891, said that he doubted the sudden stopping of the heart as Cohnheim taught; he believed that clearly the greater weight should attach to those observations in which the ligation was borne without harm, and that the stopping of the heart was not a necessary consequence of the obstruction of a large coronary branch. Hirsch in eight dogs and two apes had no sudden deaths from ligation. Bickel,[4] under Orth and Amenomiya, had a dog live nineteen days after the ligation of the descending branch of the left coronary; he killed two dogs, one on the eighth and the other on the seventeenth day after ligation. Kolster ligated smaller branches; his dogs lived, and when killed at intervals of several weeks showed the progressive changes of fibrosis of the myocardium. Imperfect technic, by which damage was done to the heart muscle and pneumothorax produced, is offered as a partial explanation, at least, for the more rapidly fatal results obtained by Cohnheim and others. Miller and Matthews[5] call attention to the better results where ether as an anesthetic is employed rather than curare or other drug. With ether they were able to ligate large branches, many of their dogs living several weeks.

Experimentally, then, sudden death, even late death, is not a necessary consequence of obstruction of even large branches, such as the descending branch of a coronary artery.

There are numerous autopsy observations, frequently with helpful clinical history, that show directly or by inference the existence of

[3] Cohnheim and Schulthess-Rechberg. Ueber die Folgen der Kranzarterienverschliessung für das Herz. Virchows Arch. f. path. Anat., 1881, lxxxv, 503.

[4] Cited by Amenomiya. (See Note 2.)

[5] Miller and Matthews. Effect on the Heart of Experimental Obstruction of the Left Coronary Artery. Arch. Int. Med., June, 1909, p. 476.

efficient anastomoses, and the ability of the heart at times to survive the obstruction of a coronary or some large branch. Some of these instructive cases may be mentioned. Pagenstecher, on account of an accident, ligated the descending branch of the left coronary artery and the patient lived five days. Thorel has seen hearts with complete obstruction of the artery, with fibrous or calcified myocardium, and yet no symptoms during life, the patient dying of some other disease. I have seen the descending branch completely occluded with an extensive fibrous area in the interventricular septum and at the apex, the latter aneurysmally dilated, where the process was clearly one of long standing. West[6] cites several cases in which at autopsy complete obstruction of one coronary was found, yet the patients had long survived this serious lesion.

Chiari, in a 32-year-old nephritic, found a *sclerosed right coronary* plugged by a thrombus, with resulting scattered patches of myomalacia cordis in the areas supplied by this artery. A portion of this thrombus had become detached and had embolically plugged the left coronary, resulting in sudden death. From the symptoms and the autopsy findings the thrombus in the right artery had formed at least two days before. The fact that the softened patches in the myocardium were scattered, with normal tissue between, and that the heart functionated fairly well until the left artery also was obstructed, leads Chiari to infer that anastomoses must exist between the right and left coronaries. Merkel[7] drew the same inference as to anastomoses from the patchy character of the lesions in the heart of a woman of 76 years, there being normal muscle between the softened areas. The left coronary was the seat of the obstruction. He also saw in a man of 37 the left coronary closed, with nourishment through the right artery. Dock[8] in a case of gradual occlusion of the right coronary artery was able to demonstrate a direct opening of the finer branches of the left coronary into the end of the right.

Spalteholz says that we all know cases of stoppage of large vessels without large infarcts resulting. Recklinghausen and Fujinami found this condition in man, as Hirsch had in dogs and monkeys, i e, smaller infarcts than the distribution of the vessel would lead one to expect. Galli saw complete closure of the entrance to the right coronary artery yet no change in the myocardium. By injection he found a round-about anastomosis between the right and left coronary arteries. Samuelson cites the case of a patient living five hours after obstruction, Huber one of a patient living several days. Aschoff and Tawara[9] saw a patient live fourteen days, "with nearly complete infarction of the parietal wall of

[6]West Tr Path Soc London 1882 xxxiv 67

[7]Merkel H Ueber den Verschluss der Kranzarterien des Herzens Festschrift fur Rosenthal Leipsic 1906

[8]Dock George Notes on the Coronary Arteries Ann Arbor 1896

[9]Aschoff and Tawara Die heutige Lehre von den pathologisch-anatomischen Grundlagen der Herzschwache Jena 1906 p 56

the left ventricle" In several cases of angina pectoris cited by Huchard[10] the patients lived many hours after the onset of the final attack, which autopsy showed was due to a thrombotic closure of an artery Osler refers to the fact that the patient may live for some time after obstruction Krehl expressly states that in man the more or less sudden occlusion of an entire coronary artery, or at least a large branch, such as the descending branch, is compatible with a continuance of life

One may conclude, therefore, from a consideration of the clinical histories of numerous cases in which there has been careful autopsy control, from animal experiments and from anatomic study, that there is no inherent reason why the stoppage of a large branch of a coronary artery, or even of a main trunk, must of necessity cause sudden death Rather may it be concluded that while sudden death often does occur, yet at times it is postponed for several hours or even days, and in some instances a complete, i e, functionally complete, recovery ensues

The clinical manifestations of coronary obstruction will evidently vary greatly, depending on the size, location and number of vessels occluded The symptoms and end-results must also be influenced by blood-pressure, by the condition of the myocardium not immediately affected by the obstruction, and by the ability of the remaining vessels properly to carry on their work, as determined by their health or disease No simple picture of the condition can, therefore, be drawn All attempts at dividing these clinical manifestations into groups must be artificial and more or less imperfect Yet such an attempt is not without value, as it enables one the better to understand the gravity of an obstructive accident, to differentiate it from other conditions presenting somewhat similar symptoms, and to employ a more rational therapy that may, to a slight extent at least, be more efficient

The variations in the results are to be accounted for in part by variations in the freedom with which anastomosing branches occur Presumably, too, symptoms will vary with the vessels or branches occluded It is conceivable that with occlusion of the right coronary the symptoms might be different from those following obstruction of the left artery, systemic edema might be a consequence of the former condition and pulmonary edema of the latter These points are, however, by no means settled either by experimental or clinical observation The condition of the remaining vessels as to patency and presence of sclerosis must play an important part in deciding how much they are capable of doing in the way of compensatory nutrition to the anemic myocardium, the strength of the heart itself, as determined, perhaps, by old valvular or myocardial disease, would also have its influence And presumably a sudden overwhelming obstruction, with comparatively normal vessels, would be followed by a profounder shock than the gradual narrowing of a lumen

[10] Huchard Traité clinique des maladies du coeur, second edition, p 360

through sclerosis which has accustomed the heart to this pathologic condition and has perhaps caused collateral circulation through neighboring or anastomosing vessels to be compensatorily increased The influence of the vessels of Thebesius is also not to be overlooked in this connection, compensatory circulation through these accessory channels may be of considerable importance in nourishing areas of heart muscle poorly supplied by sclerotic or obstructed arteries

Attempts to group these cases of coronary obstruction according to clinical manifestations must be more or less unsatisfactory, yet, imperfect as the groups are the cases may be roughly classified

One group will include cases in which death is sudden, seemingly instantaneous and perhaps painless Krehl[11] has emphasized the peculiarities of the sudden death of this type, the lack of terminal respiratory agony, of distortion of the features, of muscular contractions

A second group includes those cases in which the attack is anginal, the pain severe, the shock profound and death follows in a few minutes or several minutes at the most

In a third group may be placed non-fatal cases with mild symptoms Slight anginal attacks without the ordinary causes (such as walking), perhaps some of the stitch pains in the precordia, may well be due to obstruction of small coronary twigs Such an interpretation of these phenomena is, however, only a surmise based on the fact that other causes for the pains are lacking and that the patchy fibrosis of the myocardium that is later found at autopsy may have originated in obstruction of the sclerotic vessels, and such obstruction in small vessels may well have produced symptoms differing chiefly in degree from those caused by obstruction of larger arteries of the heart

In a fourth group are the cases in which the symptoms are severe, are distinctive enough to enable them to be recognized as cardiac, and in which the accident is usually fatal, but not immediately, and perhaps not necessarily so It is to the clinical features of this group that attention is directed in what follows

By way of introduction, I give in outline the history of a case, experience with which acutely attracted my attention to this subject

CASE 1—*History*—A man, aged 55, supposedly in good health, was seized an hour after a moderately full meal with severe pain in the lower precordial region He was nauseated and, believing that something he had just eaten had disagreed with him, he induced vomiting by tickling his throat The pain continued, however, and his physician was called, who found him cold, nauseated, with small rapid pulse, and suffering extreme pain The stomach was washed out and morphine given hypodermically The pain did not cease until three hours had passed From this time the patient remained in bed, free from pain, but the pulse continued rapid and small, and numerous râles appeared in the chest When I saw him twelve hours from the painful attack his mind was clear and

[11] Krehl Der Verschluss der Kranzarterien in Nothnagel's System xv 369

calm, a moderate cyanosis and a mild dyspnea, were present. The chest was full of fine and coarse moist râles, there was a running, feeble pulse of 140. The heart tones were very faint and there was a most startling and confusing hyperresonance over the chest, the area of heart dulness being entirely obscured. The abdomen was tympanitic. The urine was scanty, of high specific gravity, and contained a small amount of albumin and a few casts. The temperature was subnormal, later going to 99° F. Occasionally there was nausea and twice a sudden projectile vomiting of considerable fluid material. This condition remained with slight variations up to the time of death, fifty-two hours after the onset of the pain, though at one time the râles seemed nearly to have disappeared. A few hours before death, the patient described a slight pain in the heart region, but said it did not amount to much. A remarkable circumstance, and one that occasioned surprise in those who saw the patient and who realized from the almost imperceptible pulse and the feeble heart tones how weak the heart must be, was the fact that he frequently indulged in active muscular effort without evident harm. He rolled vigorously from side to side in the bed, sat suddenly bolt upright, or reached out to take things from the table near by, and once, feeling a sudden nausea, he jumped out of bed, dodged the nurse and ran into the bathroom, where he vomited, and yet seemed none the worse for these exertions.

Necropsy (Dr. Hektoen).—The heart was of normal size, but both coronary arteries were markedly sclerotic, with calcareous districts and narrowing of the lumen. A short distance from its origin the left coronary artery was completely obliterated by a red thrombus that had formed at a point of great narrowing. The wall of the left ventricle showed well-marked areas of yellowish and reddish softening, especially extensive in the interventricular septum. At the very apex the muscle was decidedly softer than elsewhere. The beginning of the aorta showed a few yellowish spots, these areas becoming less marked as the descending part was reached. An acute fibrinous pericardial deposit, which showed no bacteria in smears, was found over the left ventricle. (The pericarditis probably explains the slighter pain complained of a few hours before death.) There was marked edema of the lungs. In other respects the anatomic findings were those of health.

A colleague personally related to me the case of a man of 60 who, three days after a severe anginal seizure, felt well enough to walk on the street, though with some dyspnea. He died suddenly on the fifth day. The obstruction in the left coronary, and the muscular softening found at autopsy were similar to those in the case just described.

Since my attention has been called to this condition, I have seen five other cases that I am convinced were instances of coronary thrombosis, the patients living many hours after the accident, though no autopsy control confirms this opinion. All were men beyond 50. In all there was some evidence of peripheral arteriosclerosis, all had had previous anginal attacks. In all the final attack was described as the severest and most prolonged in the experience of the patient. Morphin alone had given relief. In all the sudden development of a weak pulse, with feeble

cardiac tones, was a striking feature, the pulse was generally rapid Dyspnea and cyanosis varied in degree Râles, moist and dry, were usually present Emphysema was present to a moderate degree in two of the five Only one patient left his bed after the attack His pulse showed great improvement as to quality and rate, though dyspnea, râles, edema of the legs, albumin, increased area of cardiac dulness, etc, showed failure of the heart muscle From the time of the seizure, i e, the time of the obstruction, to death was in one case three days, in one seven, in two twelve, and in one twenty days

One of these cases is, it seems to me, a typical one of this sort and, though necropsy is lacking, I venture to give the history

CASE 2—The patient was a man of 65, of exemplary habits His health had been good up to three years before, when he noticed at times a tight feeling in the precordia on walking For the past three months typical anginal seizures often compelled him to stop after walking two or three blocks Three days before he had had a moderately severe angina Thirty-six hours before I first saw him, in the night he made a noise, awakening his wife For a few seconds he was, perhaps, unconscious He complained of unbearably severe pain in the upper stomach region, the pain did not radiate He was nauseated and belched gas freely His physician saw him inside of twenty minutes and gave sodium bicarbonate, which was vomited The pain continuing, a hypodermic injection of morphin was necessary The patient was pale, covered with cold sweat and had a small, rapid pulse His appearance was that of collapse His distress seemed to him largely abdominal

When I saw him his color had returned and he was ruddy-cheeked He complained of extreme weakness His mind was clear There was a little cyanosis, and respiration was somewhat labored There were numerous râles in the chest The pulse was 110 and small The heart tones were faint, there was no murmur The heart was a trifle enlarged, as it had been for some years The area of cardiac flatness was decidedly small on account of overlying lung The liver dulness was but a narrow band along the costal margin, the edge of the liver could be palpated No spleen could be made out The urine contained a distinct ring of albumin and a few granular and hyaline casts There was a doubtful faint trace of bile On digalen and nitroglycerin there seemed to be some improvement in the quality of the pulse

At a second visit the condition was much the same There had been a few periods of more marked oppression in breathing, with some increase in cyanosis and weakness of the pulse

At a third visit, Oct 19, 1910, it was learned that the patient had had a bad night, with severe attacks of dyspnea The pulse had been but barely perceptible at the wrist and beat 120 to the minute At 5 A M both the physician and the patient himself had felt that death was at hand The patient had rallied, however, and when I saw him was conscious, with very feeble pulse of 110, and barely perceptible heart tones He was extremely weak Breathing was of the Cheyne-Stokes type The patient seemingly dozed during apnea, yet answered questions What I took to be a faint pericardial friction could be made out over the lower left sternal border The patient said he was not in pain He declared that he obtained relief

from swallowing orange juice, which he repeatedly sipped He remained in this condition for sixteen hours longer From the onset of his severe anginal attack to death was seven days

The instructive case of Professor Panum is described by Fraentzel[12] For a few weeks Panum had noticed dyspnea and a tight feeling on going up stairs May 1, 1885, coming home in the wind, he stopped often, and on reaching home had a sudden, severe, tearing pain in the precordia, running out to the left arm and fingers The pulse became rapid, small and irregular The patient broke out into a profuse sweat He was nauseated and induced vomiting by tickling his throat The physical findings are not accurately known The mind was clear to the last Death occurred suddenly about fourteen hours after the onset of symptoms At the necropsy both upper lobes of the lungs and the middle lobe were found emphysematous The left coronary artery was atheromatous, narrowed, and a white soft thrombus was attached to the wall The musculature of the left ventricle was degenerated and softened and had ruptured just to the left of the septum

Engelhardt[13] describes the case of a man of 54 in whom, after a thrombosis of the left coronary artery with suddenly developing gastric and abdominal symptoms, there was an illness of eight days, with fever, meteorism, vomiting, oppression, and then in a tachycardial attack rupture of the anterior wall of the left ventricle, with hemopericardium Death twelve hours after the rupture The symptom-complex resembled the picture of the abdominal-pectoral vascular crises (Pal)

A study of cases of this type shows that nearly all are in men past the middle period of life Previous attacks of angina have generally been experienced, though, as shown by my first case, the fatal thrombosis may bring on the first seizure The seizure is described by patients who have had previous experience with angina as of unusual severity, and the pain persists much longer In some instances there has been no definite radiation of the pain, as to the neck or left arm, though this may have been a feature of other anginal attacks, and the pain, as in these two cases, may be referred to the lower sternal region or definitely to the upper abdomen Cases with little or no pain have been described In Chiari's case pain is not referred to, the patient though with slow, irregular and weak pulse being out of bed The obstruction of the right coronary was, as Chiari says, "so to speak, latent" Thorel also refers to a painless case Some of Huchard's cases with obstruction did not show anginal pain Nausea and vomiting, with belching of gas, are common There may be tympany Ashy countenance, cold sweat and feeble pulse complete the picture of collapse The attention of the patient and the physician as well may, therefore, be strongly focused on the abdomen, and

[12] Fraentzel Krankheiten des Herzens, Berlin 1892 iii 51

[13] Engelhardt Ein Fall von Herzruptur, Deutsch med Wchnschr xxxvi, 1910 No 19, p 838

some serious abdominal accident be regarded as the cause of the sudden pain, nausea, collapse. The cardiac origin may be the more easily overlooked when there has been no previous typical angina, and when, as may happen (Case 1), there is no arteriosclerosis manifested peripherally and no enlargement of the heart to be made out.

Cohnheim found that in dogs the pulse after obstruction was slow. This may be so in the thrombotic obstruction of disease in man. In Hammer's[14] case the pulse dropped from 80 to 8 per minute, the patient living thirty hours from the onset of the symptoms that marked the closure of the right coronary opening. A rapid pulse is frequently seen, however. The pulse may be irregular. A striking feature has been its weakness. In two patients I have seen a rapid, thready, almost imperceptible, radial pulse, of such a quality that if met with in pneumonia or typhoid fever, it would have warranted one in presaging death within a few minutes or hours. Yet one patient lived forty hours and another four or five days with a pulse of this quality. Blood-pressure is low. The heart tones have been feeble—in fact, often startlingly feeble. Feeble contraction of the weakened, anemic heart muscle accounts for the weak pulse and the weak tones. Still another reason for the faint tones is found in the acute emphysema—*Lungenschwellung* and *Lungenstarrheit* of von Basch—by which condition the heart sounds are obscured by overlapping air-containing lungs. This also makes it difficult to map out the outlines of the heart and, coupled with the feeble apex impulse, may make such an examination for the size of the heart very unsatisfactory.

Dyspnea and cyanosis have been variable, at times much less than one would expect from the character of the accident and the quality of the heart's action. Râles, dry and moist, have been present in many cases, in some, as in my first case, largely moist, diffuse, not very large. Here there was a moderate amount of thin, frothy, slightly blood-tinged fluid expectorated, as in edema of the lungs, which condition was found at the autopsy. I mention this because some, with Cohnheim, contended that the conditions for edema would not be produced by coronary obstruction, as both right and left heart ceased beating simultaneously. Others, e.g., Samuelson, Bettelheim and Michaelis, found edema. My case shows such edema. Possibly the right heart may have remained relatively stronger than the left after the accident, and so Welch's condition for edema has been presented.

The weakness of the heart and the low blood-pressure will account for the scanty urine and the trace of albumin. A palpable liver may likewise owe its enlargement to passive congestion.

Nearly always the mind is clear—at times unusually clear—until toward the last. Some patients seem conscious, as is so common in angina, that they are face to face with death, but in none that I have seen has

[14]Hammer. Wien. med. Wchnschr., 1878, No. 5.

there been uncontrollable fear or the restlessness of fright The seriousness of the accident seemed to be realized, but there was no panic Perhaps the relief from the agony of the initial pain causes an unnatural mental calmness

General weakness has been marked in some cases, in others not One patient showed for more than a week an asthenia, comparable to that of the terminal stage of pernicious anemia or Addison's disease He hesitated to move in bed for the further reason that even turning on the side caused him the sensation as though the heart were giving out Even slight movement caused some pain His case is representative of the type of status anginosus Obrastzow regards this as the usual manifestation of coronary thrombosis My experience shows that such obstruction may be followed by a complete cessation of pain for hours, and even to the time of death Some of these patients of the latter type will, if permitted, move freely or even get out of bed

The occurrence of a serofibrinous exudate over the area of myocardial softening, with roughening of the pericardium, has been noted in several instances This may explain a later precordial distress, as in Case 1 A fine pericardial friction, therefore, occurring several hours or a few days after the initial pain, may be confirmatory evidence of coronary obstruction Osler[15] concluded, in one of his cases of angina, that the attack was probably associated with acute infarct of the ventricle, "as a pericardial rub was detected the next day" Dock[8] recognized this pericarditis *intra vitam* in one of his cases and found it post mortem over the softened area In one of Leyden's cases in which the patient lived five days from the onset of symptoms of dizziness, faintness, small pulse, there was found myomalacia cordis, and especially at the apex, where a softened area reached the surface, there was pericarditis, cloudy fluid was in the pericardial sac This was almost certainly a case of coronary obstruction, though the occluding lesion is not described This pericarditis is in keeping with some of the experimental work on lower animals, e g, that of Bickel, who in his dogs killed nineteen and seventy days after ligation found localized pericarditic adhesions over the area representing the myocardial softening

Death is the result in nearly all of these cases Yet it may be delayed for many days More than this, there is, as has been shown by reference to experimental work, no intrinsic reason why some patients with obstruction of even large branches of the coronary artery may not recover Experimental animals sometimes do And as already said, mild cases must occur, and one cannot pretend to say where the dividing line should be drawn between the mild obstruction of a coronary branch, whose recovery means a few fibrous patches in the myocardium, and the more serious one that in a few days is to lead to rupture of the heart or is

[15] Osler Lumleian Lectures on Angina Pectoris, Lancet London March 12 and 26, and April 9, 1910

to produce an extensive weakened fibrous area that will ultimately yield in cardiac aneurysm. Death may then be caused by rupture, by sudden asystole, or by gradual giving out of the weakened heart muscle—by "ingravescent systole," as Balfour[16] styles it—a mode of death occupying from half an hour to a week, illustrated by one of his cases in which death occurred one week after the obstruction, which was found at post mortem. In one instance in which I believe the anginal seizure was thrombotic a dilatation of the heart, with orthopnea, dropsy, etc., followed the seizure. Death here was, as in cardiac failure, from other causes. Some of the dogs of Miller and Matthews died in this way several weeks after the ligation of the coronary. In cases in which the heart slowly wears out in the course of a few days, Cheyne-Stokes respiration, general asthenia, urinary scantiness, with mental apathy and exhaustion may be present.

Emphasis ought to be laid on the resemblance of some of these cases to surgical accidents. The sudden onset with pain over the lower sternal and epigastric region, the nausea and vomiting, the tympany, the feeble pulse, ashy color, cold sweat and other signs of collapse make one think of such conditions as gall-bladder disease, acute hemorrhagic pancreatitis, perforation of gastric or duodenal ulcer, hemorrhage into the adrenal capsule, etc. The dyspnea, hyperresonant thorax, obscured heart tones, may suggest pneumothorax or diaphragmatic hernia. In my first case, while the diagnosis made was that of cardiac accident, there were so many disquieting features that surgical counsel was called to make sure that some surgical accident, such as those enumerated, had not been overlooked. Details as to differential diagnosis need not be given. Where there is arteriosclerosis, enlarged heart, a history of previous angina, typical radiation of the pain to the neck and arm, the diagnosis will not be so difficult as where these suggestive aids are lacking. The bilateral character of the emphysema, the persistence of breath sounds, often with râles, the failure of the heart to be dislocated, will help exclude pneumothorax and diaphragmatic hernia. The absence of blood from the vomitus, the absence of peritonitic tenderness, a study of the temperature, the leukocytes, etc., will help in excluding subdiaphragmatic accidents.

Obrastzow[17] calls particular attention to this resemblance to surgical accidents which my own experience corroborates. Engelhardt's case also illustrates this point.

If these cases are recognized, the importance of absolute rest in bed for several days is clear. It would also seem to be far wiser to use digitalis, strophanthus or their congeners than to follow the routine practice of

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[16] Balfour. Clinical Lectures on Diseases of the Heart, Edition 3, 1898, pp. 316 and 328.

[17] Obrastzow and Straschesko. Zur Kenntniss der Thrombose der Koronararterien des Herzens. Ztschr. f. klin. Med., 1910, lxxi, 116.

giving nitroglycerin or allied drugs The hope for the damaged myocardium lies in the direction of securing a supply of blood through friendly neighboring vessels so as to restore so far as possible its functional integrity Digitalis or strophanthus by increasing the force of the heart's beat, would tend to help in this direction more than the nitrites The prejudice against digitalis in cases in which the myocardium is weak is only partially grounded in fact Clinical experience shows this remedy of great value in angina, and especially in cases of angina with low blood pressure, and these obstructive cases come under this head The timely use of this remedy may occasionally in such cases save life Quick results should also be sought by using it hypodermically or intravenously Other quickly acting heart remedies would also be of service

OLIVER WENDELL HOLMES

(Courtesy Medical Classics)

The Stethoscope Song
A Professional Ballad

By

OLIVER WENDELL HOLMES

There was a young man in Boston town,
He bought him a stethoscope nice and new,
All mounted and finished and polished down,
With an ivory cap and a stopper too

It happened a spider within did crawl,
And spun him a web of ample size,
Wherein there chanced one day to fall
A couple of very imprudent flies

The first was a bottle fly, big and blue,
The second was smaller, and thin and long,
So there was a concert between the two,
Like an octave flute and a tavern gong

Now being from Paris but recently,
This fine young man would show his skill,
And so they gave him, his hand to try,
A hospital patient extremely ill

Some said his liver was short of bile,
And some that his heart was oversize,
While some kept arguing, all the while,
He was crammed with tubercles up to his eyes

This fine young man then up stepped he,
And all the doctors made a pause,
Said he, The man must die, you see,
By the fifty seventh of Louis's laws

But since the case is a desperate one,
To explore his chest it may be well,
For if he should die and it were not done
You know the autopsy would not tell

Then out his stethoscope he took,
And on it placed his curious ear,
Mon Dieu! said he with a knowing look,
Why, here is a sound that's mighty queer!

The *bourdonnement* is very clear,—
Amphoric buzzing, as I'm alive!
Five doctors took their turn to hear,
Amphoric buzzing, said all the five

There's empyema beyond a doubt,
We'll plunge a trocar in his side
The diagnosis was made out,—
They tapped the patient, so he died

Now such as hate new fashioned toys
Began to look extremely glum,
They said that rattles were made for boys,
And vowed that his buzzing was all a hum

There was an old lady had long been sick,
And what was the matter none did know
Her pulse was slow, though her tongue was quick,
To her this knowing youth must go

So there the nice old lady sat,
With phials and boxes all in a row,
She asked the young doctor what he was at,
To thump her and tumble her ruffles so

Now, when the stethoscope came out,
The flies began to buzz and whiz,
Oh, ho! the matter is clear, no doubt,
An aneurism there plainly is

The *bruit de rape* and the *bruit de scie*
And the *bruit de diable* are all combined,
How happy Bouillaud would be,
If he a case like this could find!

Now, when the neighboring doctors found
A case so rare had been described,
They every day her ribs did pound
In squads of twenty, so she died

Then six young damsels, slight and frail,
Received this kind young doctor's care,
They all were getting slim and pale,
And short of breath in mounting stairs

They all made rhymes with "sighs" and "skies,"
And loathed their puddings and buttered rolls,
And dieted, much to their friends' surprise,
On pickles and pencils and chalk and coals

So fast their little hearts did bound,
The frightened insects buzzed the more,
So over all their chests he found
The *râle sifflant* and the *râle sonore*

He shook his head There's grave disease,—
I greatly fear you all must die,
A slight post mortem, if you would please,
Surviving friends would gratify

The six young damsels wept aloud,
Which so prevailed on six young men
That each his honest love avowed
Whereat they all got well again

This poor young man was all aghast!
The price of stethoscopes came down,
And so he was reduced at last
To practice in a country town

The doctors being very sore,
A stethoscope they did devise
That had a rammer to clear the bore,
With a knob at the end to kill the flies

Now use your ears, all you that can,
But don't forget to mind your eyes,
Or you may be cheated, like this young man,
By a couple of silly, abnormal flies

THE CORRELATION OF THESE CLASSICS WITH OTHER CONTEMPORARY HISTORIC EVENTS

THE CORRELATION OF THESE CLASSICS WITH OTHER CONTEMPORARY HISTORIC EVENTS

YEAR	AUTHOR	CONTRIBUTION INCLUDED IN THIS VOLUME	COUNTRY	CONTEMPORARY HISTORIC EVENT
1628	William Harvey	On the motion of the heart and blood	England	The third parliament of Charles I. Passage of the Petition of Rights
1640	Pierre Gassendi	A note on the demonstration of the foramen ovale in the adult	France	Reign of Louis XIII. The rise of culture under Cardinal Richelieu
1661	Marcello Malpighi	Demonstration of the capillary circulation	Italy	A year after the restoration of the English Monarchy
1664	Niels Stensen	On the muscular nature of the heart	Denmark	Reign of Frederick III, six years after the Danish Swedish War
1705	William Cowper	Description of aortic insufficiency	England	Second parliament of Anne. Whigs in majority
1708	Antony van Leeuwenhoek	His conception of "that motion which we call the pulse"	Holland	Holland at war with France and Spain (1702-13)
1733	Stephen Hales	Early experiments on hemodynamics	England	Reign of George II. Two years after the treaty of Vienna
1749	Jean Baptiste de Senac	Treatment of "rebellious palpitation" with quinine	France	Reign of Louis XV. Struggle between the church, parliament and crown
1755	Albrecht von Haller	Description of calcification of the heart and pericardium	Switzerland	Period of tranquility in the Swiss cantons
1761	John Baptist Morgagni	Description of certain pathologic lesions of the heart	Italy	Naples and Sicily were governed by Ferdinand
1761	Leopold Auenbrugger	The introduction of percussion	Austria	Seven Years' War, Silesia ceded to Prussia
1772	William Heberden	The first accurate description of angina pectoris	England	Decision of Court of King's Bench that slavery cannot exist in Great Britain
1785	William Withering	The first treatise on digitalis	England	Two years after the recognition of the independence of the United States of America

1788	Matthew Baillie	Description of congenital dextrocardia with complete situs transversus	Scotland	The period of the French Revolution
1794	John Hunter	Classic description of his anginal attacks	England	Treaty with the United States of America (Jay's treaty)
1806	Jean Nicolas Corvisart	Description of the signs of contraction of the orifices of the heart, etc	France	France, under Napoleon, at war with Prussia and Russia
1812	William Charles Wells	The earliest account relating rheumatic fever with heart disease	United States and England	The War of 1812 between the United States and Great Britain
1818	John Cheyne	Description of that periodic type of breathing later to be known as Cheyne Stokes respiration	Scotland	The reign of George III, three years after the battle of Waterloo
1819	Rene T H Laennec	The introduction of the stethoscope and auscultation	France	Two years before the death of Napoleon I
1825	Caleb Parry	Description of the circulatory phenomena of exophthalmic goiter	England	Financial panic and failure of banks
1827	Robert Adams	Description of heart block	Ireland	Battle of Navarino, destruction of the Turkish fleet
1831	James Hope	Description of cardiac asthma, stenosis of the pulmonary valves, and cardiac neurosis	England	British Association for the Advancement of Science held its first meeting
1832	Dominic John Corrigan	Description of the pulse in aortic insufficiency	Ireland	Irish reform act passed
1835	Jean Baptiste Bouillaud	The pathology of endocarditis	France	Death of Dupuytren, the famous French surgeon
1846	William Stokes	Description of heart block	Ireland	Failure of the potato crop in Ireland resulting in famine
1852	William Senhouse Kirkes	Discussion of emboli from intracardiac ganglia	England	Disraeli was Chancellor of the Exchequer
1854	William Stokes	Description of that periodic type of breathing later to be known as Cheyne Stokes respiration	Ireland	Reciprocity treaty between the United States and Great Britain

YEAR	AUTHOR	CONTRIBUTION INCLUDED IN THIS VOLUME	COUNTRY	CONTEMPORARY HISTORIC EVENT
1861	Paul Louis Duroziez	Description of the femoral murmurs in aortic insufficiency	France	Mexican Expedition
1862	Austin Flint	Description of the murmur later to be known as the Austin Flint murmur	United States	One year following Lincoln's inauguration as President Civil War one year old
1867	Pierre Carl E Potain	Description of the pulsations in the jugular veins	France	The Luxembourg question
1867	Thomas Lauder Brunton	Introduction of amyl nitrite in the treatment of angina pectoris	England	The formation of the Dominion of Canada
1868	Heinrich Quincke	Demonstration of the capillary and venous pulse	Germany	A year before the formal opening of the Suez Canal
1870	Samuel Wilks	Description of bacterial endocarditis	England	Death of Charles Dickens
1872	Ludwig Traube	Demonstration of pulsus alternans	Germany	A year after the close of the Franco Prussian War
1876	William Richard Gowers	Description of the oculi fundi in hypertension	England	Victoria proclaimed Empress of India
1877	Julius Cohnheim	Description of paradoxical embolism	Germany	Turco Russian War
1879	Henri Roger	Description of the murmur of patency of the interventricular septum	France	Jules Grevy President of the Third Republic of France
1879	William Murrell	The introduction of nitroglycerin in the treatment of angina pectoris	England	Strike of London engineers against reduction of wages
1885	Pierre Carl E Potain	Discussion of gallop rhythm	France	Death of Victor Hugo
1887	Augustus D Waller	Demonstration of a method of leading the action currents of the heart from the surface of the heart	England	Triple Alliance renewed

1887	John A MacWilliam	Experimental production of various cardiac arrhythmias	England	Nordenfelt's boat submarine successfully tried at Southampton
1888	Graham Steell	Description of the murmur of high pressure in the pulmonary artery, later to be known as the Graham Steell murmur	England	Death of Matthew Arnold
1888	Etienne Louis A Fallot	Description of the "maladie bleue," later to be known as the tetralogy of Fallot	France	67,000,000 francs voted for the defense of Brest, Toulon, and Cherbourg
1893	Wilhelm His, Jr	Demonstration of the auriculoventricular bundle (bundle of His)	Germany	Tariff controversy between Germany and Russia in progress
1896	Francis H Williams	Early study of fluoroscopy of the heart and aorta	United States	Election of President McKinley
1897	William H Broadbent	Classic description of adherent pericarditis	England	Workmen's Compensation Act approved
1903	Willem Einthoven	Introduction of the string galvanometer (electrocardiograph)	Holland	Gift of $1,500,000 by Andrew Carnegie for a temple of peace for the permanent court of arbitration at The Hague, reported
1904	Ludwig Aschoff	Classic description of rheumatic myocarditis (Aschoff nodule)	Germany	Arbitration treaty between Great Britain and Germany
1907	Arthur Keith and Martin Flack	Demonstration of the sinoauricular node and its function as the pacemaker of the heart	England	Death of Sir Michael Foster, famed physiologist
1908	James Mackenzie	Description of auricular fibrillation	Scotland	Wireless telegraph extended between Montreal and London
1909	William Osler	Description of the skin nodules in subacute bacterial endocarditis, later to be known as Osler nodes	Canada, United States, England	Peary's expedition to the North Pole
1912	James B Herrick	Classic description of coronary thrombosis	United States	New Mexico and Arizona admitted to the Union

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